# **C**onditionally **A**ctive **B**iologics: Transforming Cancer Therapy

Corporate Presentation

March 2026

bioatla

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# Selective and Targeted CAB Technology Widens Therapeutic Window

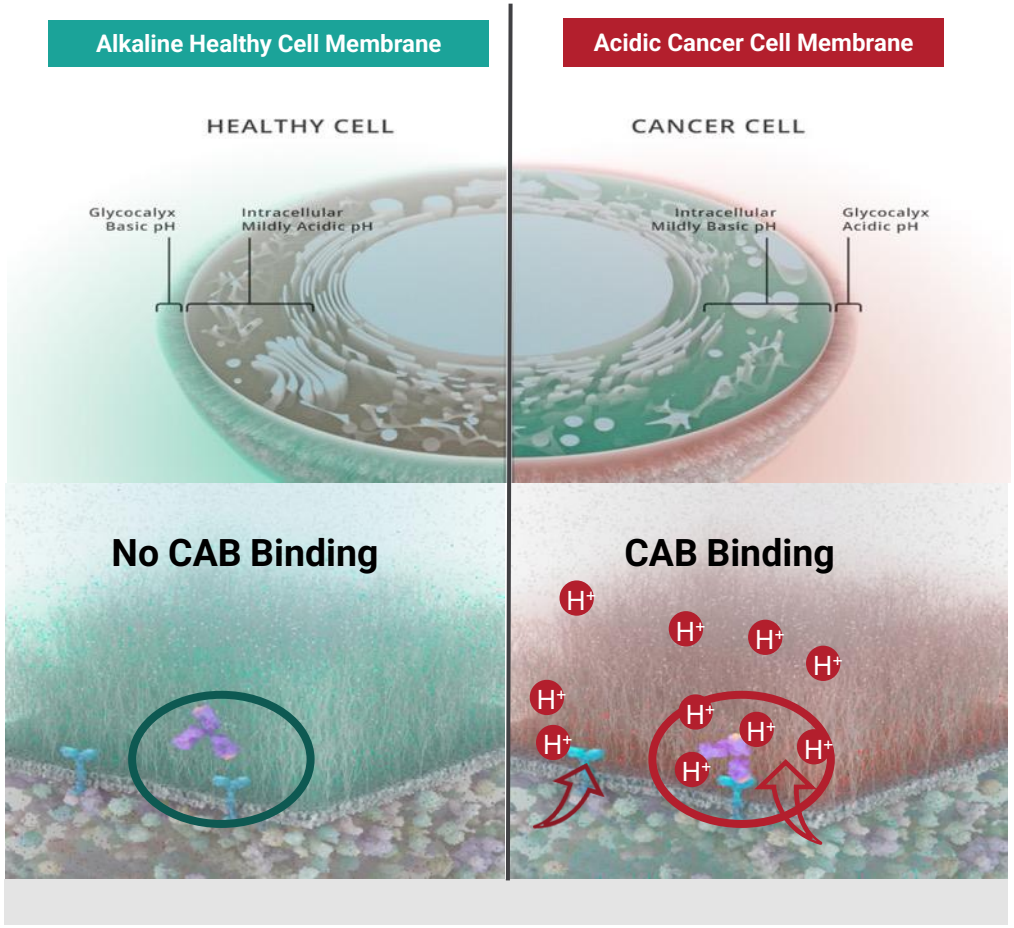
## Thus has the potential to enhance clinical outcomes in multiple tumor types

BioAtla discovered that acidic pH at the cancer cell surface unveils binding sites that are shielded at normal pH of healthy cells

BioAtla invented CAB technology, creating antibodies that bind ***only*** to these unveiled sites on cancer cells

CAB binding region is not masked or caged and thus different from prodrugs that require irreversible enzymatic cleavage to become activated

CAB antibodies have the potential for increased efficacy with improved safety relative to traditional antibodies




Chang, H.W., Frey, G., Liu, H., Xing, C., Steinman, L, Boyle, B.J., & Short, J.M. (2021) PNAS 118(9): 1-10, Suppl. 1-19.

# CAB Platform Technology Summary

- All cancer cells are acidic (pH5.3-pH6.7)
  - The most acidic regions are oxygenated, not anaerobic
  - Acidity is a result of the need for precursor molecules from glycolysis for continuous cell replication
  - Cancer cells use acidity to promote metastasis and defend against immune response
- CAB mechanism
  - Leverages naturally occurring, negatively charged molecules (*e.g.* bicarbonate, hydrogen sulfide) to differentiate between targets on cancer cells versus normal cells
  - These physiological molecules underpin the CAB mechanism and are referred to as Protein-associated Chemical Switches (PaCS)™
  - In normal tissues, PaCS shield epitopes, so CAB antibodies cannot bind. In contrast, cancer cells produce H+ ions that remove PaCS molecules from the epitopes, enabling cancer-specific binding.

# Myths vs Facts of pH Therapies in Cancer

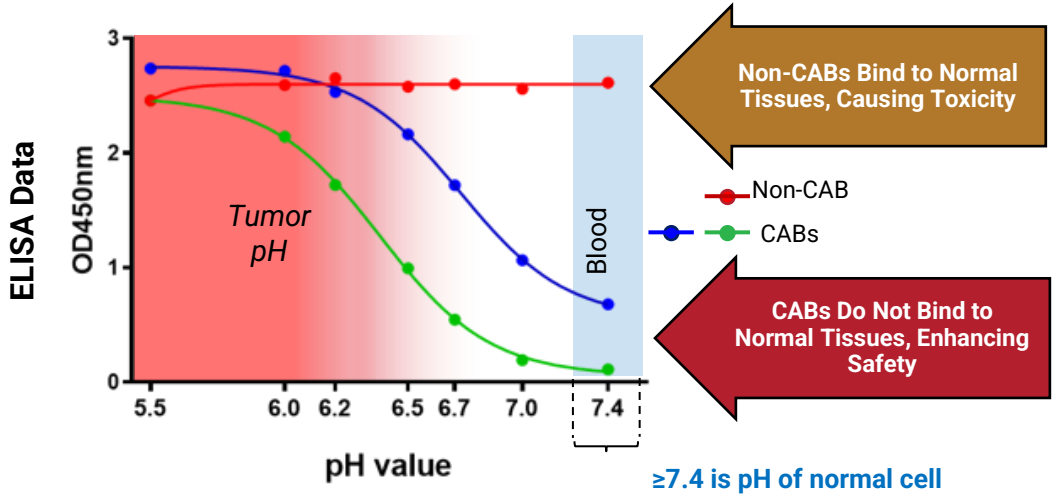
## BioAtla's Solution: Conditionally Active Biologics (CAB)

| Myth | Fact |
|---|---|
| Not all tumors are acidic | ALL tumors and cancer cells are acidic (*i.e.* pH5.3-6.7). |
| Cancer cells are ATP limited | Cancer cells are NOT generally ATP deficient but are limited in other precursor molecules whose synthesis depends upon glycolysis. |
| pH technology will miss tumor cells because tumor pH is variable | While tumor degree of pH acidity can vary, CABs are designed to bind any cancer cell at or below a predetermined pH. |
| Tumor size influences the acidic environment so pH technology will not work | • Larger tumors with larger anaerobic regions are not necessarily more acidic since oxygenated regions have higher acidity due to the higher concentration of hydrogen ions from rapid glycolysis.<br>• Cancer cells – as opposed to the average pH of a tumor – are more acidic, especially at the membrane of the cancer cell. |

# CAB Antibodies Bind Selectively and Reversibly Based on the Tumor Microenvironment (TME)

## Enhancing exposure and reducing toxicity

### CABs Bind Selectively in the Lower pH TME



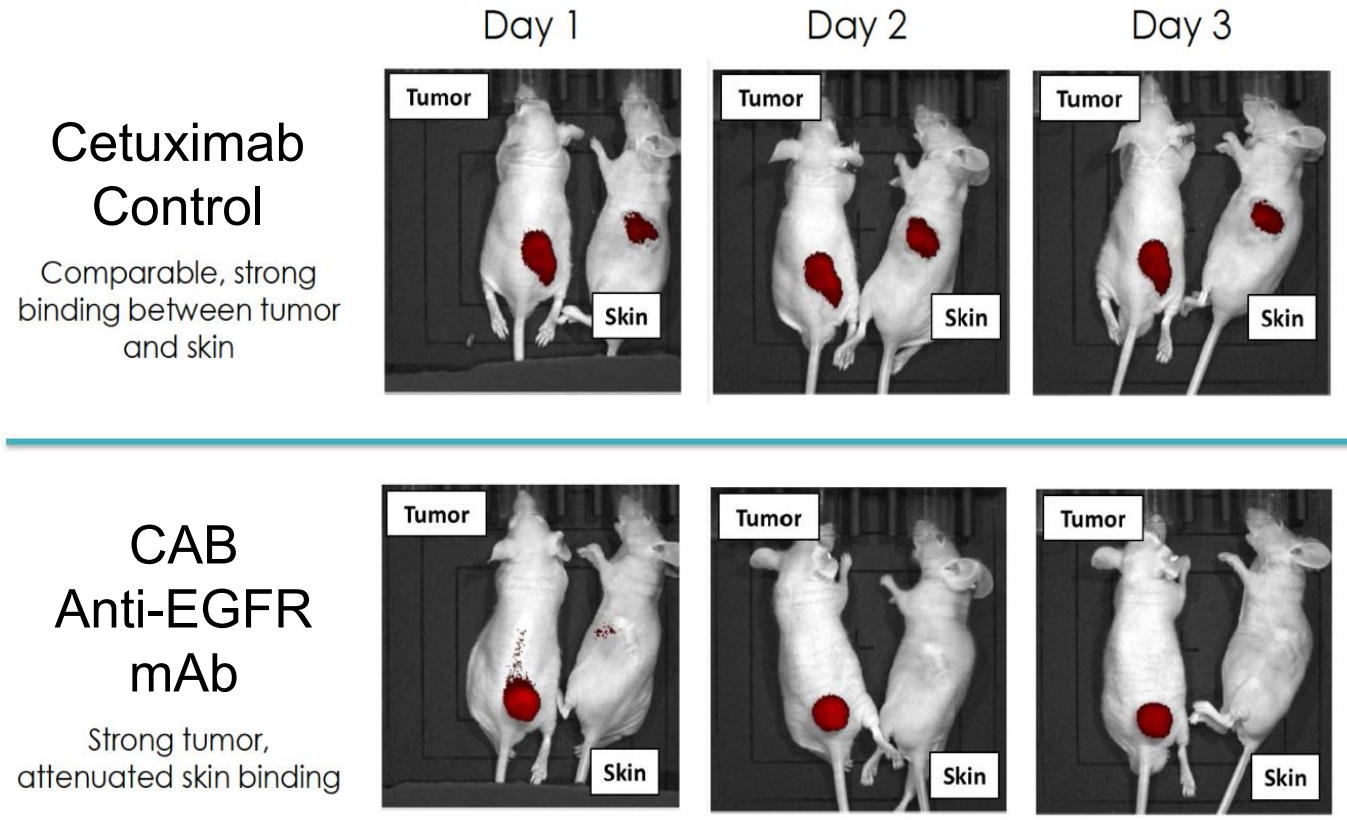

Selective Binding → **Focused Tumor Killing** → Normal Cells Preserved

Note: OD450nm = optical density measurements using a microplate reader with a 450nm filter; TME = Tumor Micro Environment; mABs = monoclonal antibodies; Data above based on non-human primate studies

# CAB Technology Eliminates On-target, Off-tumor Binding

## Thus, widening the therapeutic index by 12.6-fold over cetuximab

**Enhanced Tumor Selectivity**
**(12.6-fold increase in TI)**




*PET scan images*

# Conditional Binding Approaches: Prodrug vs BioAtla's CAB Platform Technology

| Feature | Proteolytic cleavage of prodrug | Conditionally Active Biologic (CAB) |
|---|---|---|
| **Mechanism of Action** | **Mask** obscuring antibody binding site is cleaved by tumor-associated proteases | Biologic is engineered to bind **only in TME at low pH*** |
| **Trigger Type** | **Enzymes** overexpressed in tumors | **TME** (*i.e.*, low pH) |
| **Bioengineering** | **Addition of foreign sequence** | **No foreign sequences** |
| **Activation Precision** | **Requires activation**; dependent on expression and enzymatic efficiency | **No activation required** – maximizes potency |
| **Risk of Off-Target Effects** | Irreversible activation - **can bind** target in **normal tissues** | Reversible binding - **will not bind** target in **normal tissues** |

TME = Tumor microenvironment
*Warburg effect

# Preclinical Evidence Summary

- Preclinical evidence of CAB selectivity
  - Differential EGFR tumor vs. skin binding (12.6-fold improved TI)
  - AXL-ADC reduced TMDD yielding
    - Increased $T_{1/2}$ and exposure in NHP (>2-fold increase in $T_{1/2}$)
    - Reduced liver enzymes (<10% of non-CAB ALT levels)
  - EpCAM DualCAB TCE maintained efficacy with highly reduced toxicity
    - MTD not reached in NHP
    - >100-fold improvement in TI
  - B7H3 Dual CAB TCE; target expression associated with high acidity via hyper-glycolysis
    - MTD not reached in NHP
    - Encouraging safety profile compared to other B7H3 TCEs in development
  - CTLA4 reduced peripheral immune response while maintaining efficacy
    - Maintains efficacy at same dose, while enabling higher and extended dosing
    - Significant reduction in colitis in NHP compared to ipi
    - MTD not reached at 30 mg/kg in NHP
    - Selective reduction of activated T cells in the periphery or normal tissues

CAB, Conditionally Active Biologics; TI, Therapeutic Index; $T_{1/2}$, Half-life; TMDD, Target—Mediated Drug Disposition; NHP, Non-Human Primate; ALT, Alanine Aminotransferase; TCE, T-Cell Engager; MTD, Maximally Tolerated Dose; ipi, ipilimumab

# Clinical Evidence Summary

- Clinical evidence of CAB selectivity
  - AXL-ADC good risk/benefit ratio
    - Two non-CAB AXL-targeting ADCs terminated in P1 due to toxicity
    - Potent and durable response with differentiated OS in mKRAS NSCLC and sarcoma patients
  - ROR2-ADC good risk/benefit ratio
    - Good tolerability with only 7% treatment-related discontinuation rate
    - Potent and durable response in SCCHN patients
  - EpCAM DualCAB TCE
    - Non-CAB EpCAM TCE (BiTE) terminated in P1
    - Most advanced EpCAM TCE in the clinic showing tumor-reduction, ongoing in P1
    - MTD not yet reached
  - CTLA4 I/O enables higher and prolonged dosing with reduced immune-meditated AEs
    - Maintains PK and efficacy at similar dose, while enabling more intensive dosing
    - MTD not reached at 14.3 mg/kg
    - Extended dosing (>2x over ipi) and at higher doses
    - Reduced grade 3 AEs such as colitis even at higher doses

**CABs demonstrate universal clinical improvement in TI and enable therapeutic development "undruggable" targets**

# Key Advantages of the CAB Platform

## Widening The Therapeutic Index

- ✓ **Conditional and reversible binding increases clinical activity and improves safety**
- ✓ **Not dependent on enzymatic activation for selective binding**
- ✓ **Enhances pharmacologic properties**
- ✓ **Broadly applicable to antibody formats including ADCs, Bispecific TCEs, CAR-Ts and other proteins**
- ✓ **Provides ability to create new therapies and combinations against targets that had previously been limited due to toxicity**

# Clinical and Preclinical Pipeline of Differentiated CAB Assets Across Multiple Solid Tumors

| | CAB Program | Target | Indications | IND Enabling Pre-Clinical | Phase 1 Clinical | Phase 2 Clinical | Phase 3 Clinical |
|---|---|---|---|---|---|---|---|
| CAB-Bispecific TCE | BA3182 | EpCAM x CD3 | Adenocarcinomas | | | | |
| | CT-202 (Out licensed to Context Therapeutics) | Nectin-4 x CD3 | Solid Tumors | | | | |
| | BA3142 | B7H3 x CD3 | Solid Tumors | | | | |
| | BA3241 | Trop2 x CD3 | Solid Tumors | | | | |
| | BA3311 | EGFR x CD3 | Solid Tumors | | | | |
| CAB-ADCs | BA3021 *Ozuriftamab Vedotin* | ROR2 | 2L+ OPSCC | | | | |
| | BA3011 *Mecbotamab Vedotin* | AXL | NSCLC Sarcoma | | | | |
| | BA3361 | Nectin-4 | Solid Tumors | | | | |
| CAB-I/O | BA3071 *Evalstotug* | CTLA-4 | 1L and 2L Melanoma | | | | |

IND, investigational new drug; OPSCC, oropharyngeal squamous cell carcinoma; NSCLC, Non-small Cell Lung Cancer; TCE, T cell engager

# BA3182 (Dual CAB EpCAM x CD3 Bispecific T-Cell Engager): Adenocarcinoma

# Why EpCAM (Epithelial Cell Adhesion Molecule) As a Target?

## Targeting EpCAM has potential to serve over 1 Million patients (potential Pan-cancer drug)

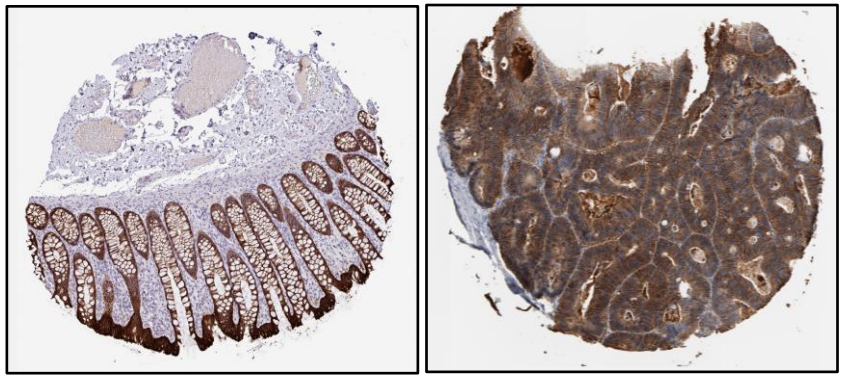
| | Estimated Number of New Cancer Cases in 2025 | EpCAM Expression (TIS 1 to 12)[2] |
|---|---|---|
| Breast Cancer | 319,750 | 81% |
| Prostate Cancer | 313,780 | 99% |
| Lung Cancer | 226,650 | 93% NSCLC<br>80% SCLC |
| Colon Cancer | 154,270 | 100% |
| Pancreatic Cancer | 67,440 | 99% |
| Thyroid Cancer | 44,020 | 97% |
| Ovarian | 20,890 | 92% |
| Gallbladder & other biliary | 12,610 | 97% |

[1]Siegel RL, Kratzer TB, Giaquinto AN, Sung H, Jemal A. Cancer statistics, 2025. *CA Cancer J Clin*. 2025.
[2]G. Spizzo, et al. J Clin Pathol 2011;64:415e420.

**Challenges of targeting EpCAM**

All normal epithelia express EpCAM which with traditional antibodies would lead to on-target, off-tumor toxicities

CABs are essential for targeting EpCAM



**Normal Colon** **Colon Cancer**

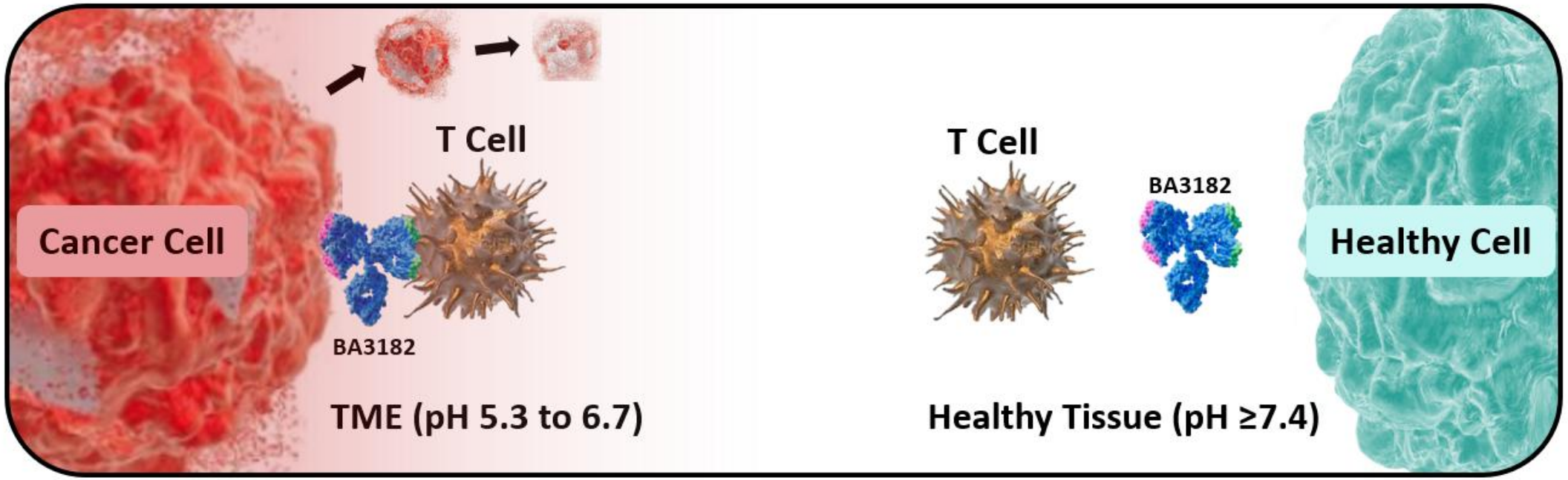
EpCAM IHC

# Proposed Mechanism of Action of BA3182, a Dual-CAB EpCAM x CD3 bispecific T-cell Engager

## CAB-TCEs redirect all T cells in the TME to attack cancer cells, but not in healthy tissues

**Dual-conditional binding technology drives selective ablation of cancer cells in the acidic TME**

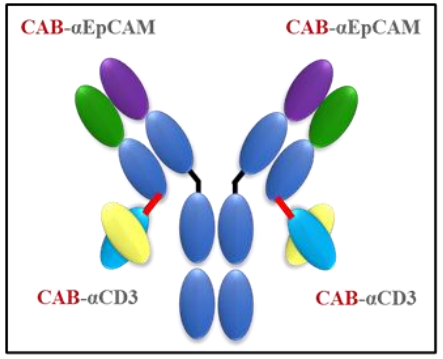
**No binding in healthy tissue, reducing CRS and on-target, off-tumor toxicities**




Mabs. 2024 Mar 6;16(1):2322562

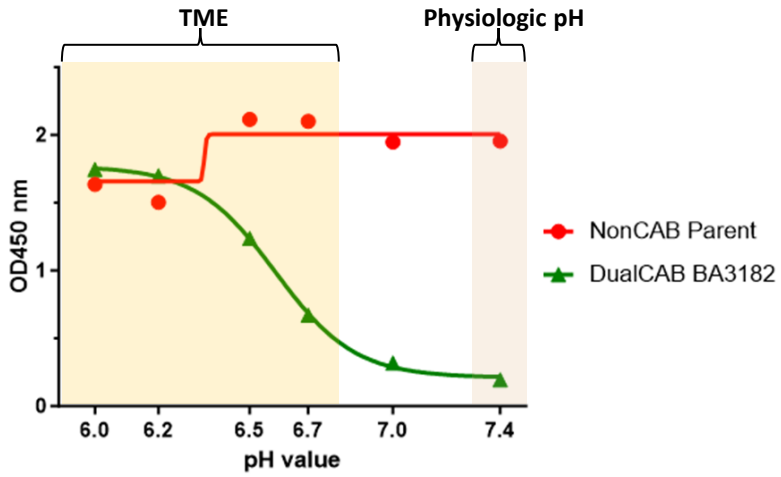
# BA3182 – First Dual-CAB T-cell Engager Targeting EpCAM

## Potent Lysis of EpCAM Positive Cancer Cells by BA3182



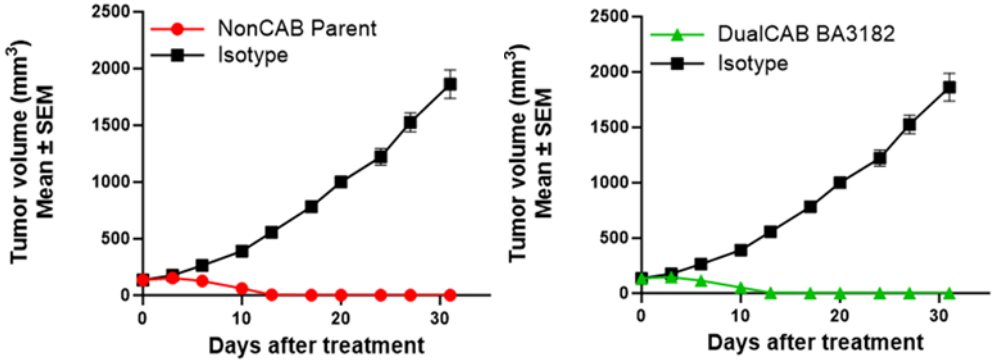

Tetravalent T-cell engager EpCAM*

### pH Range ELISA




Highly reduced binding to both targets at normal physiological pH

### HCT116 CDX Model



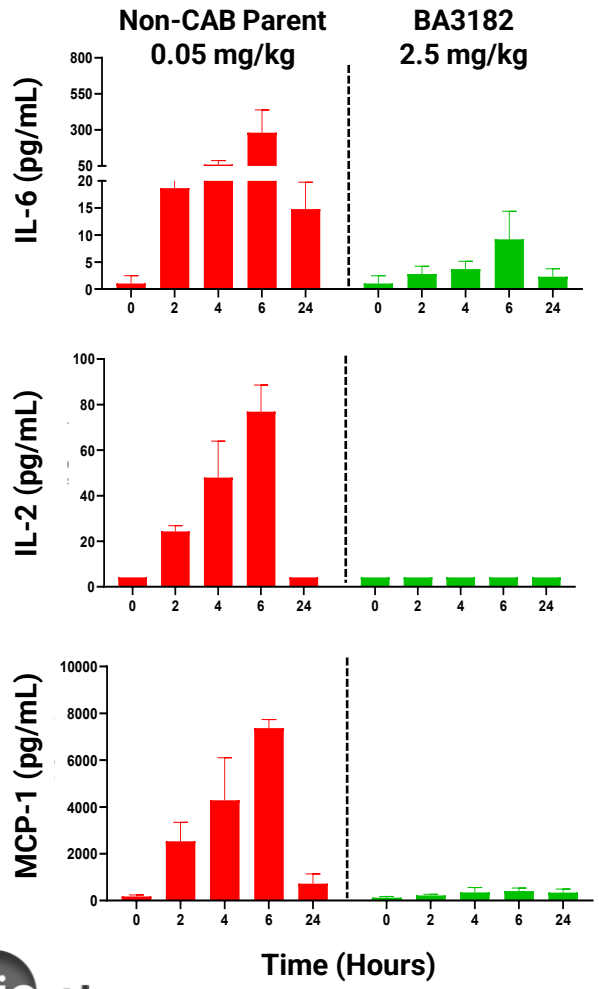

Similar efficacy in mouse xenograft models compared to Non-CAB parent molecule

*(2+1) functional format (only one anti-CD3scFv can engage a T cell based on steric hindrance)

Mabs. 2024 Mar 6;16(1):2322562

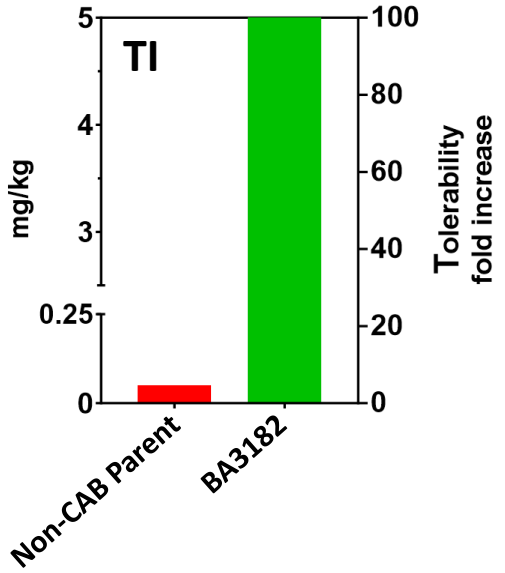
# CAB-EpCAM-TCE, BA3182, is Well Tolerated at High Doses in Non-human Primates

## Cytokine Levels Associated with Toxicity




Low cytokine levels with DualCAB vs Non-CAB Parent even at significantly higher doses

## Increase in Safety and Tolerability




| Test Article | Non-CAB Parent | BA3182 |
|---|---|---|
| Dose | 0.05 mg/kg | 5 mg/kg |
| Clinical Outcome | All Euthanized on Day 8 | No Clinical Findings |

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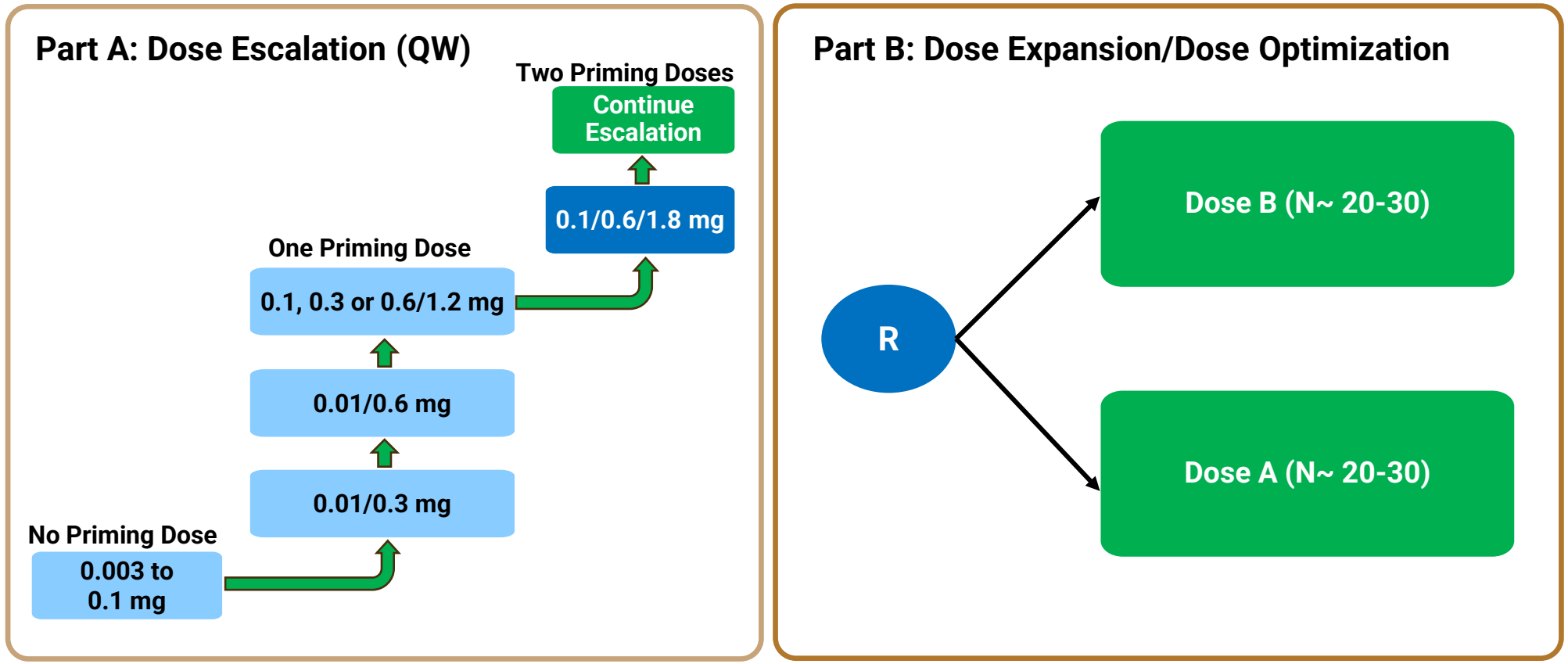
# Phase 1 Dose Escalation Study of BA3182 in Advanced Adenocarcinomas

## Trial ongoing; evaluating various dosing and treatment schedules




Treatment notes:

- Prophylactic acetaminophen and diphenhydramine delivered prior to all doses and prophylactic tocilizumab (no corticosteroids) given prior to Cycle 1 Day 1 treatment dose
- Post treatment ondansetron guided for nausea
- Ongoing weekly treatment dosing continued after DLT observation interval concluded

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# BA3182 Phase 1 Patient Demographics

## Patients dosed SC per protocol as of September 10, 2025: N=35

| Patient Characteristic | N=35 |
|---|---|
| Age, mean (SD), y | 57 (10) |
| Male (n, %) | 19 (54) |
| Female (n, %) | 16 (46) |
| ECOG performance status | |
| 0 (n, %) | 24 (69) |
| 1 (n, %) | 11 (31) |
| Presence of liver metastases (n, %) | 22 (63) |

| Carcinoma Type (n, %) | N=35 | Median # of prior treatments |
|---|---|---|
| Adenoid Cystic Carcinoma | 1 (3) | 2 |
| Appendiceal | 1 (3) | 2 |
| Cholangiocarcinoma | 2 (6) | 1 |
| Colorectal | 22 (63) | 4 |
| Gallbladder | 1 (3) | 3 |
| Ovarian | 1 (3) | 8 |
| Pancreas | 7 (20) | 3 |

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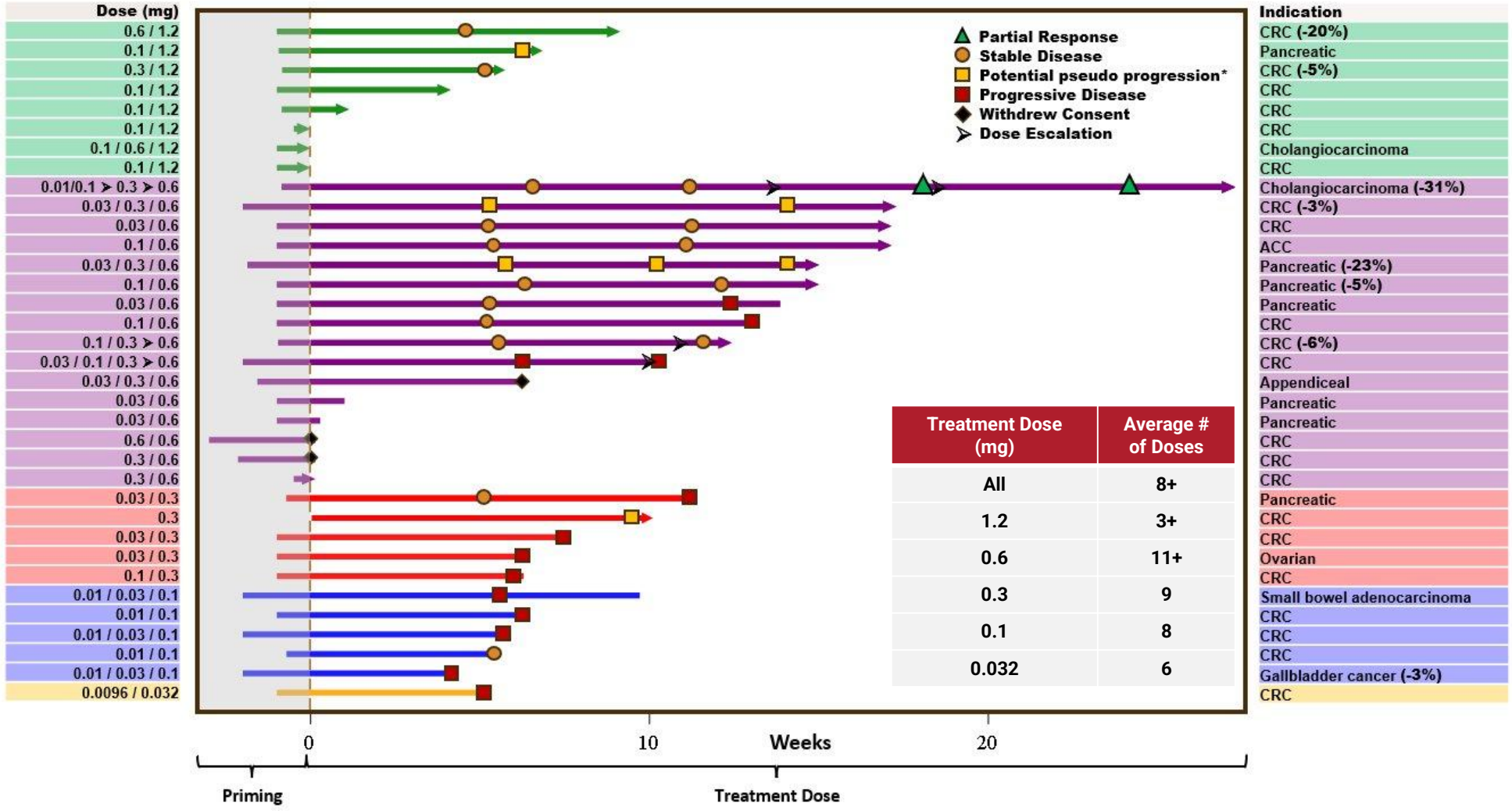
Data cut as of 10Sep25

# BA3182 Preliminary Prolonged Tumor Control With Increasing Doses




| Treatment Dose (mg) | Average # of Doses |
|---|---|
| All | 8+ |
| 1.2 | 3+ |
| 0.6 | 11+ |
| 0.3 | 9 |
| 0.1 | 8 |
| 0.032 | 6 |

*Pts with unconfirmed PD were treated beyond initial progression per protocol when pseudo-progression was suspected

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Live Data cut as of 10Sep2025

# BA3182 Achieved Objective Tumor Size Reductions Across Multiple Tumor Types

## Preliminary assessment of anti-tumor activity among pts receiving treatment doses of ≥0.6 mg (N=24)

- 14 CRC pts: among 7 pts who had one scan – 5 achieved SD at 1st scan; among the 7 pts without scans –2 withdrew consent before the 1st scan and 5 are pending 1st scan
- 6 pancreatic cancer pts: 2 pts had SD at 1st scan and 2 pts continued treatment beyond potential pseudo-progression
- 2 cholangiocarcinoma pts: 1 pt achieved a confirmed PR and 1 pt is pending first scan
- 1 ACC pt experienced SD
- Among pts treated with ≥0.6 mg , 9/10 pts achieved SD at a higher rate and remained on active treatment for prolonged intervals, generally longer than those who received lower doses

CRC, colorectal cancer; SD, stable disease; PR, partial response; ACC, adenoid cystic carcinoma

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Live Data cut as of 10Sep2025

# BA3182 Adverse Events Generally Transient and Readily Manageable

## Preliminary safety of subcutaneous dosing

| Characteristic | N=35 (n, %) |
|---|---|
| Any Adverse Events (AEs) | 29 (83) |
| Related AEs of Grade 3-4 | 14 (40) |
| Related AEs of Grade 3 hepatic analytes[1] | 11 (31) |
| Related AEs of Grade 4 hepatic analytes[1] | 1 (3) |
| Related AEs of Grade 3-4 non-febrile neutropenia[2] | 2 (6) |
| Related AEs of Grade 3 excluding hepatic analytes[1] and non-febrile neutropenia[2,4] | 3 (9) |
| Any related serious AEs[3] | 5 (14) |
| Related CRS of any grade (per ASTCT grading) | 2 (6) |
| Related AEs leading to death | 0 |
| Related AEs leading to treatment discontinuation | 1 (3) |

[1]Early, transient elevation of hepatic analytes: AST, ALT, bilirubin, and/or alkaline phosphatase
[2]Non-febrile, transient neutropenia, possibly related to tocilizumab
[3]G2 pancreatitis, G2 atrial fibrillation, G3 diarrhea, G3 acute kidney injury, and G2 CRS
[4]G3 diarrhea; G3 diarrhea/lymphocyte count decrease; G3 acute kidney injury/white blood cell count decrease

| TRAE >10% (N=35) | TRAE Any (n, %) | TRAE G3+ (n, %) |
|---|---|---|
| Alanine aminotransferase increased* | 15 (42.9) | 7 (20.0) |
| Aspartate aminotransferase increased* | 15 (42.9) | 9 (25.7) |
| Nausea | 14 (40.0) | 0 |
| Injection site reaction | 12 (34.3) | 0 |
| Diarrhea | 11 (31.4) | 2 (5.7) |
| Fatigue | 9 (25.7) | 0 |
| Blood bilirubin increased* | 8 (22.9) | 0 |
| Neutrophil count decreased | 7 (20.0) | 2 (5.7) |
| Blood alkaline phosphatase increased* | 6 (17.1) | 1 (2.9) |
| Decreased appetite | 6 (17.1) | 0 |
| Dysgeusia | 5 (14.3) | 0 |
| Vomiting | 5 (14.3) | 0 |
| Abdominal pain | 4 (11.4) | 0 |
| Constipation | 4 (11.4) | 0 |

*Transient laboratory changes; resolved

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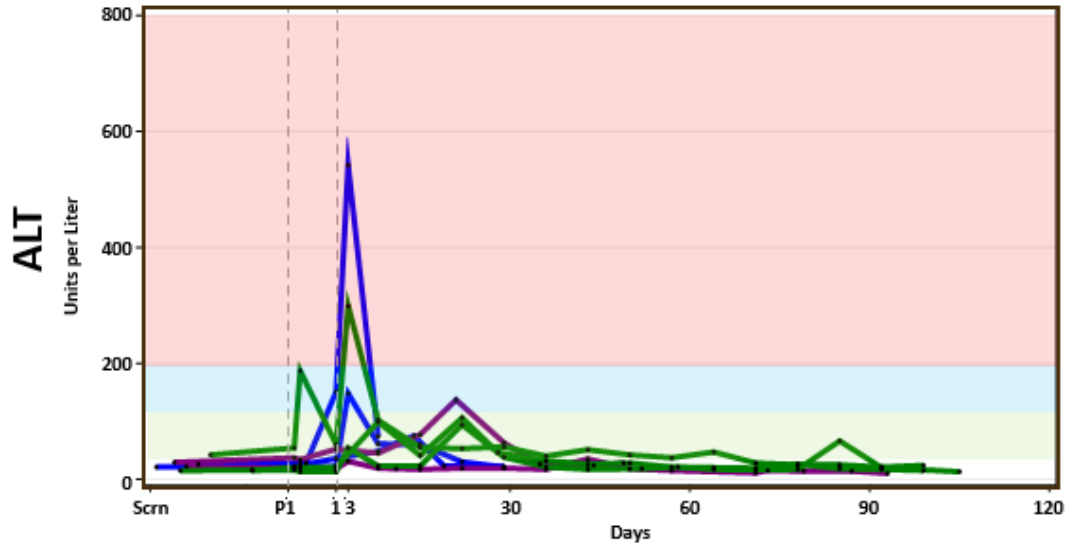
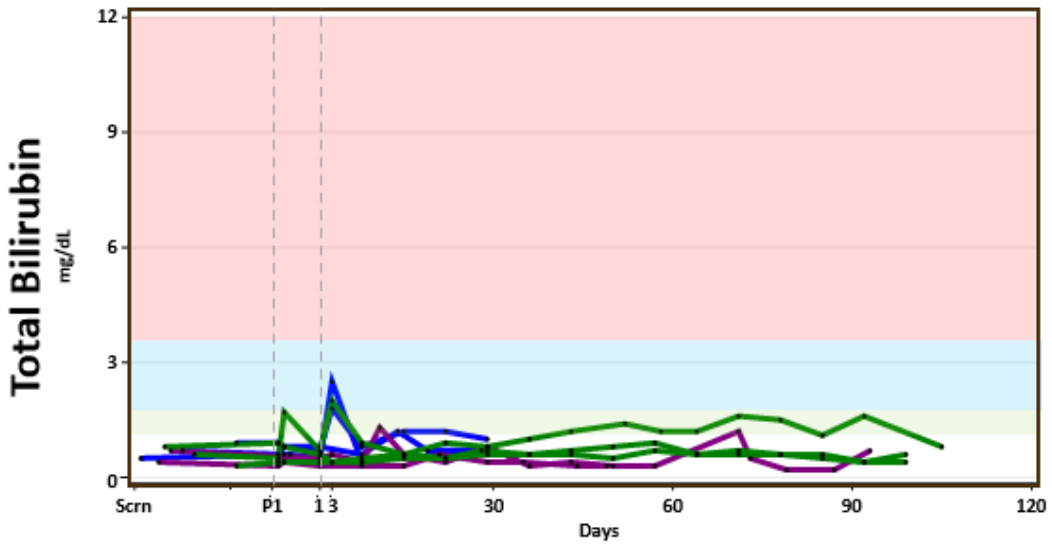
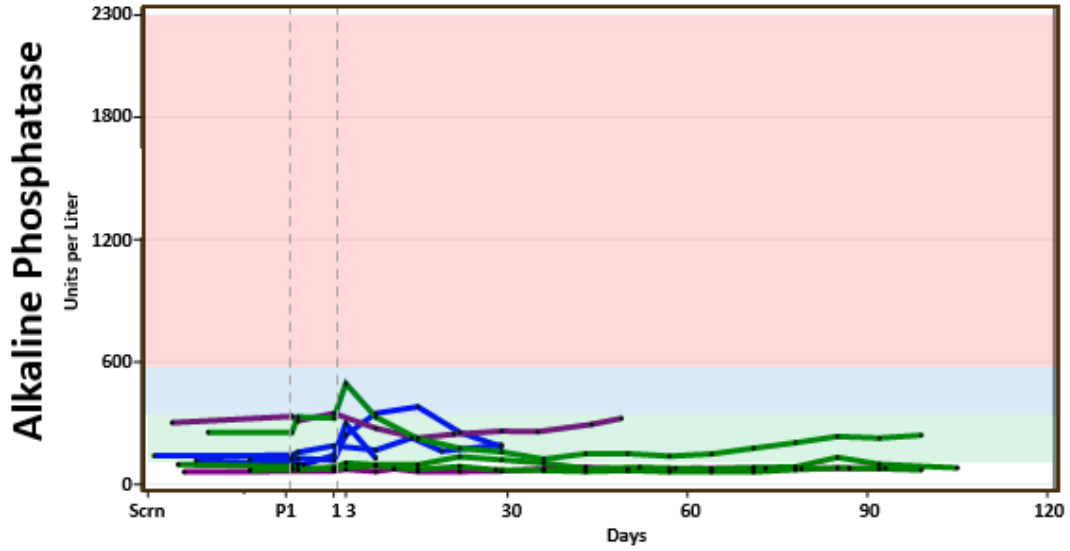
Data cut as of 10Sep25

# Early, Transient Increases in Hepatic Analytes Resolved with Continued Dosing

## Patients primed with 0.1 mg prior to treatment dose (N=9; treatment doses: 0.3, 0.6, and 1.2 mg)




**Summary of hepatic analyte changes:**

- Early, transient, and asymptomatic
- Enabling on-time weekly treatment dosing
- Consistent with cholestasis (not on target toxicity)

**Grade 3**
**Grade 2**
**Grade 1**
**Normal Range**

**0.1 / 1.2 mg (n=4)**
**0.1 / 0.6 mg (n=3)**
**0.1 / 0.3 mg (n=2)**

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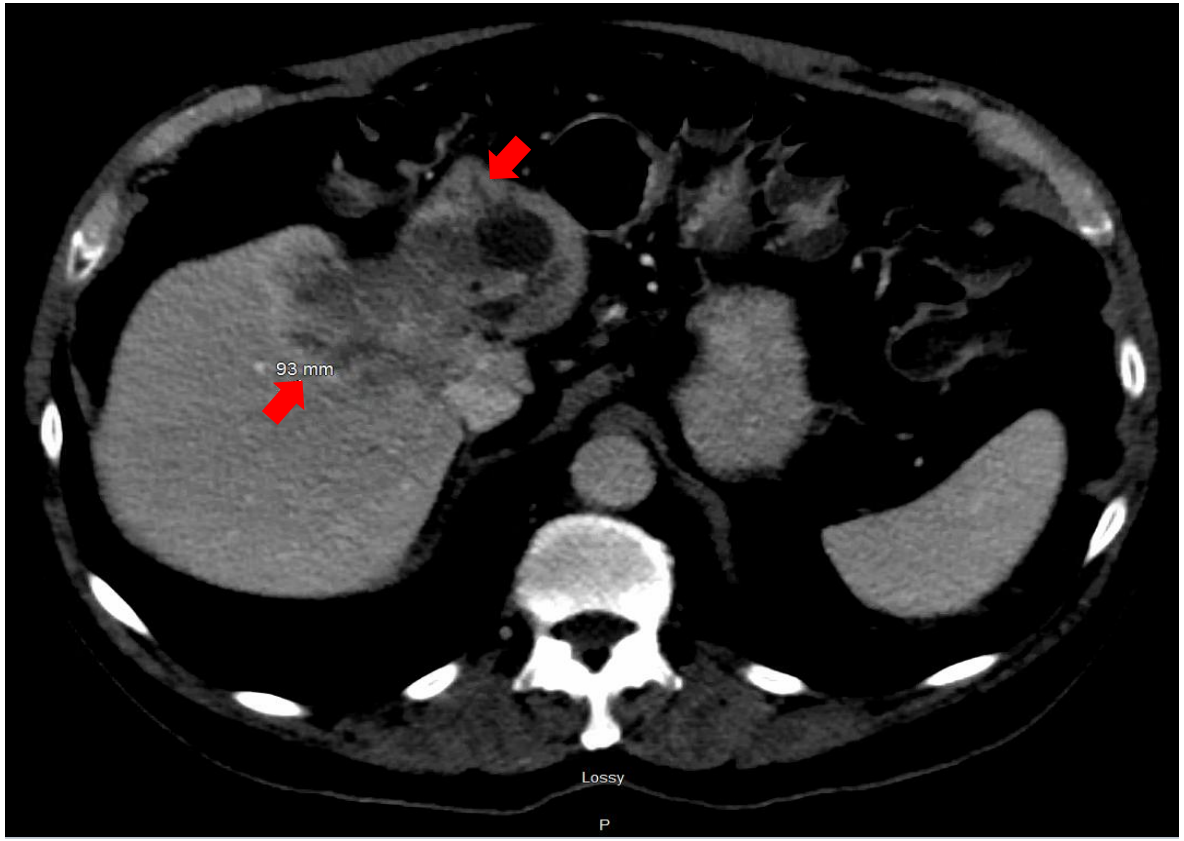
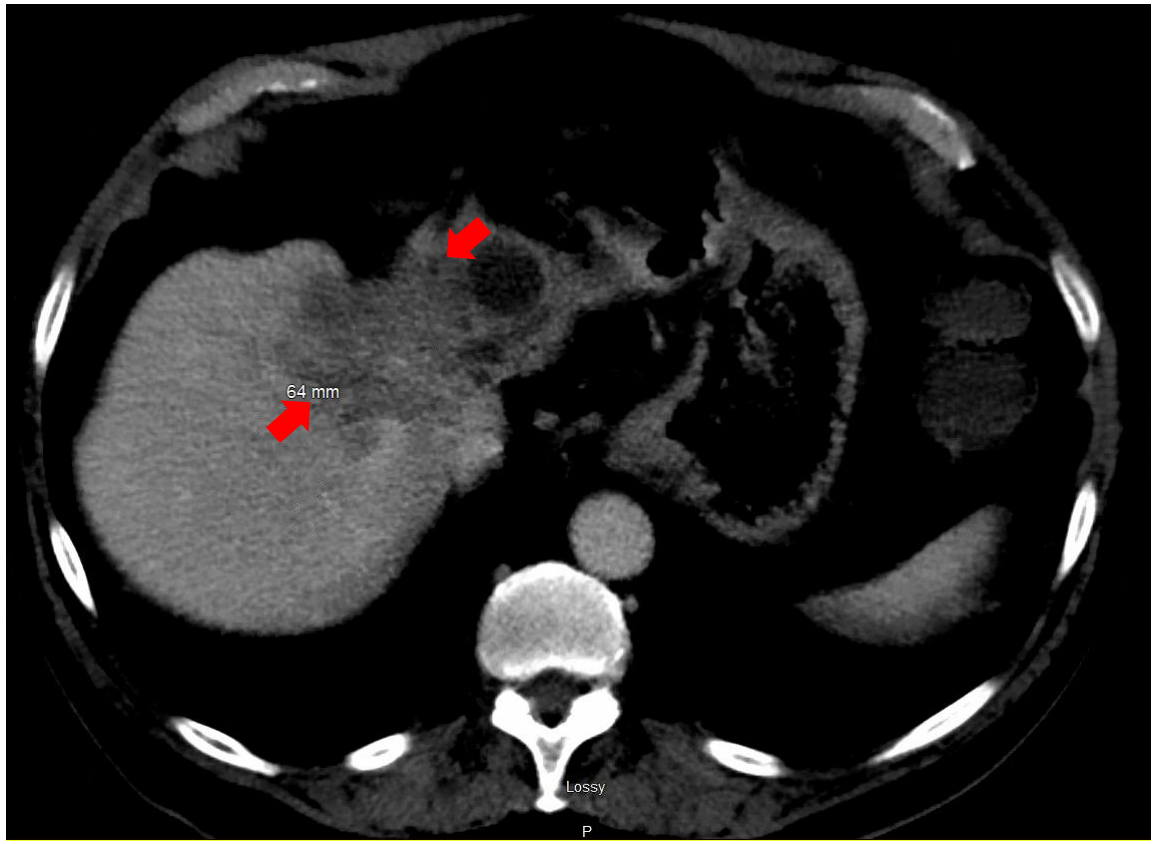
Live Data cut as of 10Sep2025

# Confirmed Partial Response (31% Tumor Reduction) BA3182 at 0.6 mg in Patient with Intrahepatic Cholangiocarcinoma without Progression for >6 months

## 71-year-old male with stage IV cholangiocarcinoma previously treated on clinical trial with gemcitabine, cisplatin, durvalumab, and investigational agent.

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Data cut as of 10Sep25

# BA3182 Summary / Upcoming Milestones

- BA3182, a dual-conditionally binding CAB-EpCAM x CAB-CD3 T-cell engager continues to demonstrate a manageable safety profile with preliminary evidence of antitumor activity
- Early cytokine increases appear to cause brief, reversible cholestasis – consistent with tumor-selective targeting
- BA3182 treatment achieved an ongoing, confirmed partial response and multiple patients have experienced prolonged tumor control; currently testing dose levels up to 1.8mg and dose varying frequencies
- Dose escalation read out anticipated 1H’26

# Ozuriftamab Vedotin (CAB-ROR2-ADC): Oropharyngeal Squamous Cell Carcinoma (OPSCC)

# Significant Opportunity for Oz-V in OPSCC

## Rapidly Growing Patient Population

- OPSCC incidence is increasing, largely due to HPV-driven disease[1,2]
- Up to 80% of OPSCC cases in the United States caused by HPV infection[3]
- By 2030, OPSCC will be the most common subtype of head and neck cancer in the US[1,2]

## Potential to Address Significant Unmet Need

- OPSCC poorly served by SOC, including EGFR inhibitors[4-10]
- Oz-V has a compelling and differentiated profile in HPV+ OPSCC

## Large Commercial Opportunity with Potential to Expand

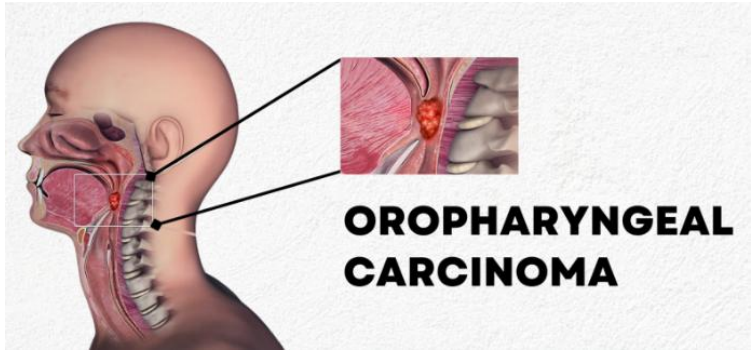
- $800mm WW peak sales projections in 2L+ OPSCC[11]
- Total WW OPSCC market value projected to be ~$3Bn by 2032[12]
- Total HPV+ solid tumors (ie, cervical) worldwide market valued at >$7Bn[13]

[1]Med Sci (Basel). 2023 Jun 13;11(2):42.; [2]Oral Oncol. 2021 Apr:115:105177.; [3]Dela J Public Health. 2023 Apr 22;9(1):26-28. [4]N Engl J Med 2016;375:1856-1867. [5]Journal of Clinical Oncology 2018; 36(15): 1551-1558. [6]Cohen E, et al. (2019) Lancet 393, 156–167. [7]British Journal of Cancer (2018) 119:153–159; https://doi.org/10.1038/s41416-018-0131-9. [8]2008 Jun 15;112(12):2710-9. doi: 10.1002/cncr.23442, [9]Erbitux USPI accessed 2024., [10]INTERLINK-1: Phase 3 study of cetuximab ± monalizumab /Volume 34, Supplement 2S554-S555 October 2023., [11]Internal BioAtla projections.; [12]https://www.coherentmi.com/industry-reports/oropharyngeal-cancer-market; [13]Cervical Cancer Therapeutics Market Size, Demand, Report to 2033; 23-cervix-uteri-fact-sheet.pdf

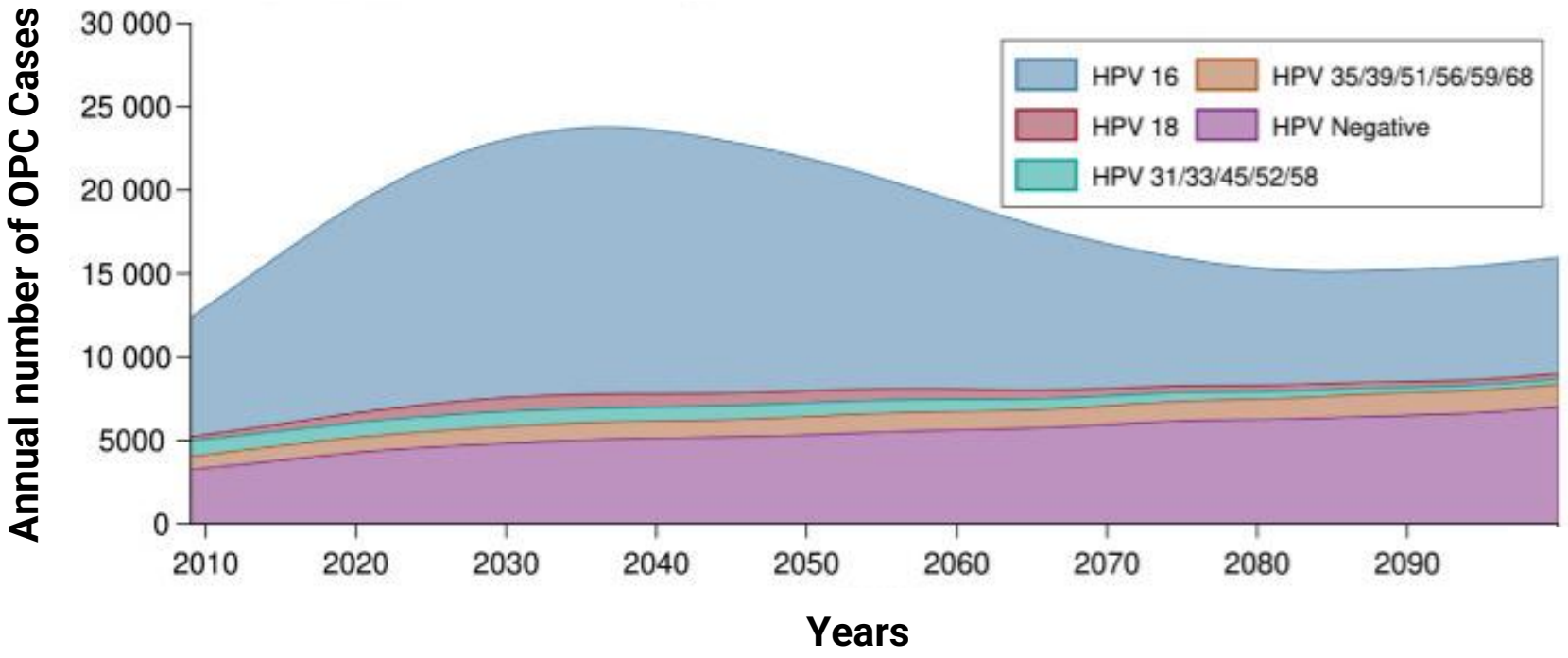
OPSCC, Oropharyngeal Squamous Cell Carcinoma; SOC, Standard of Care; WW, worldwide

# OPSCC Incidence is Increasing

## Proportion of new HPV+ OPSCC diagnoses now approach 80% in the US




**Oropharyngeal Cancer Burden by Infection Status**

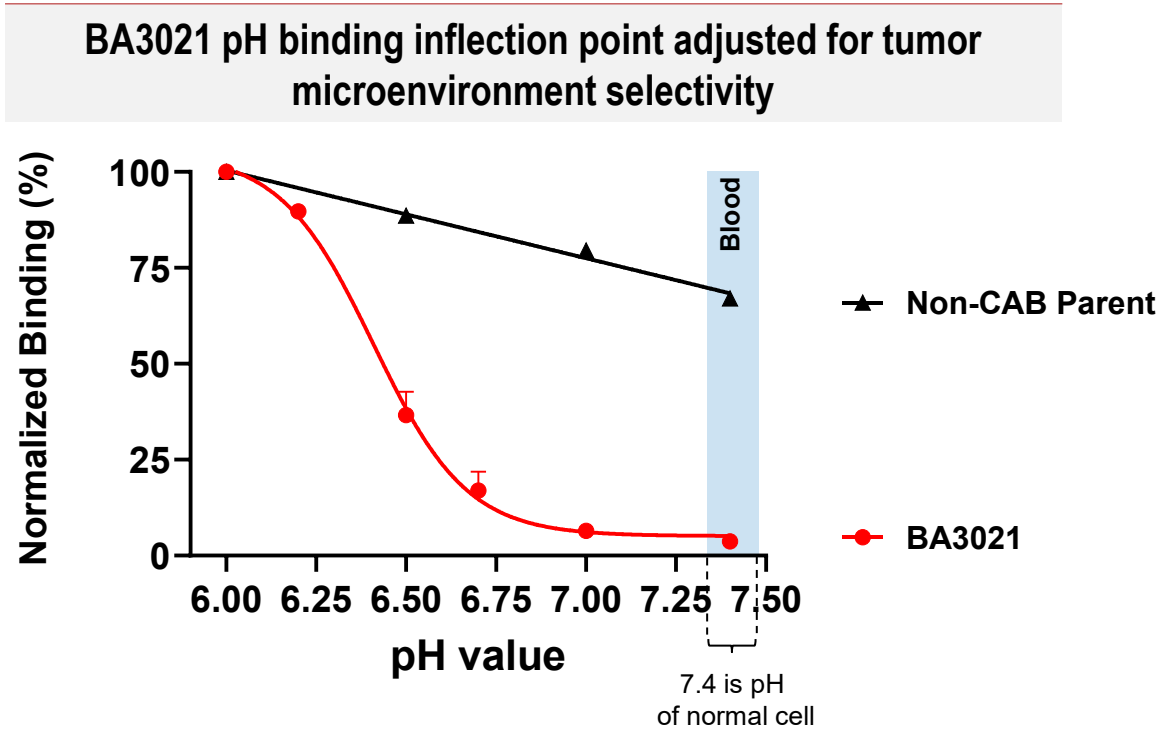


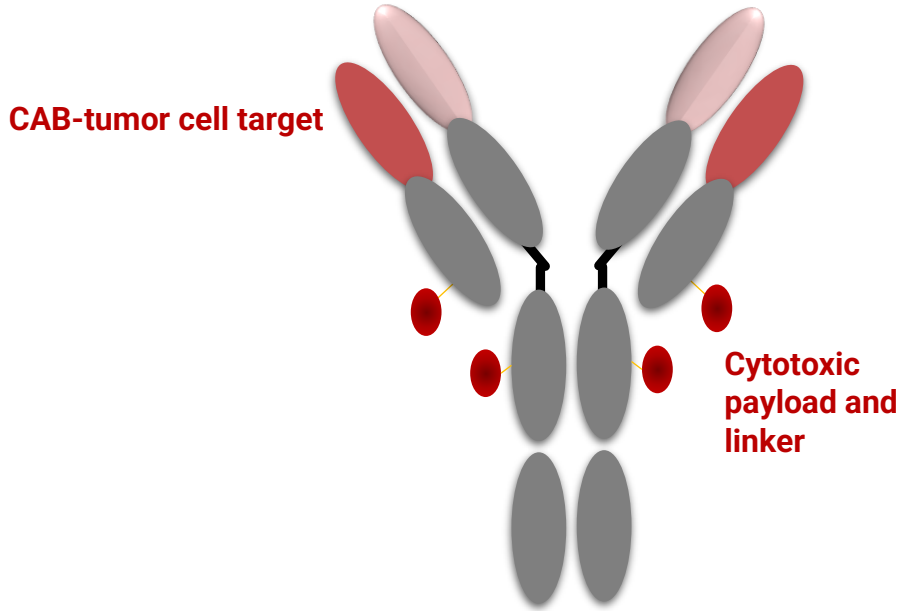

- Oropharyngeal cancer has now surpassed cervical cancer as the most common HPV-related malignancy
- More than 21,000 U.S. cases yearly compared to nearly 12,000 cervical cancer cases*

Damgacioglu H, Sonawane K, Chhatwal J, et al. Long-term impact of HPV vaccination and COVID-19 pandemic on oropharyngeal cancer incidence and burden among men in the USA: A modeling Study. The Lancet Regional Health – Americas. 2022;8:100143.
*CDC report.

# Ozuriftamab Vedotin (Oz-V): CAB-ROR2-ADC

## ROR2 is expressed in a variety of tumor types, with overexpression associated with metastasis, tumor resistance to chemotherapy, and poor prognosis




- MMAE-containing ADC (DAR4) with cleavable linker
- Humanized anti-ROR2 (N-terminal) IgG1
- ~2nM affinity (pH 6)
- MMAE-containing ADC (DAR4) with cleavable linker
- Epitope in Ig loop region

# ROR2 Overexpression is Driven by HPV E6 / E7 Oncoproteins

## 80% of OPSCC cases in the United States caused by HPV infection

- HPV driven cancers
  - **Highest** ROR2 expression among SCCHN
  - HPV E6 and E7 oncoproteins **drive ROR2 overexpression**
  - ROR2 overexpression results in increased proliferation and invasiveness
- Oz-V conditionally and selectively eliminates ROR2-expressing cells

HPV infection drives ROR2 overexpression

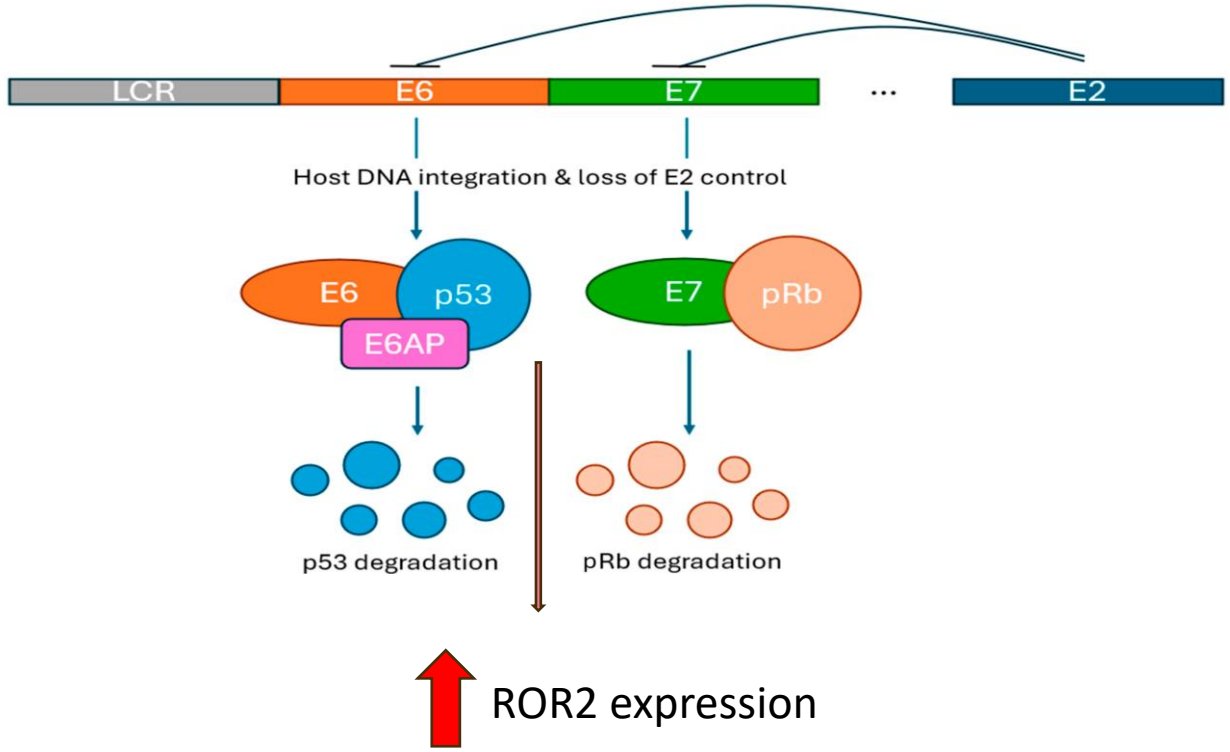



Figure adapted from Z. Lu, et al. Cancers. 2024, 16, 3474.

Carrero, et al. Oncogene (2019) 38:3551–3568; https://doi.org/10.1038/s41388-018-0659-4
Avincsal, et al. Oncol Rep. 2021 Jul;46(1):148. doi: 10.3892/or.2021.8099. Epub 2021 Jun 3. PMID: 34080643.

# Demographics and Baseline Clinical Characteristics

## HPV p16 positive and all patients

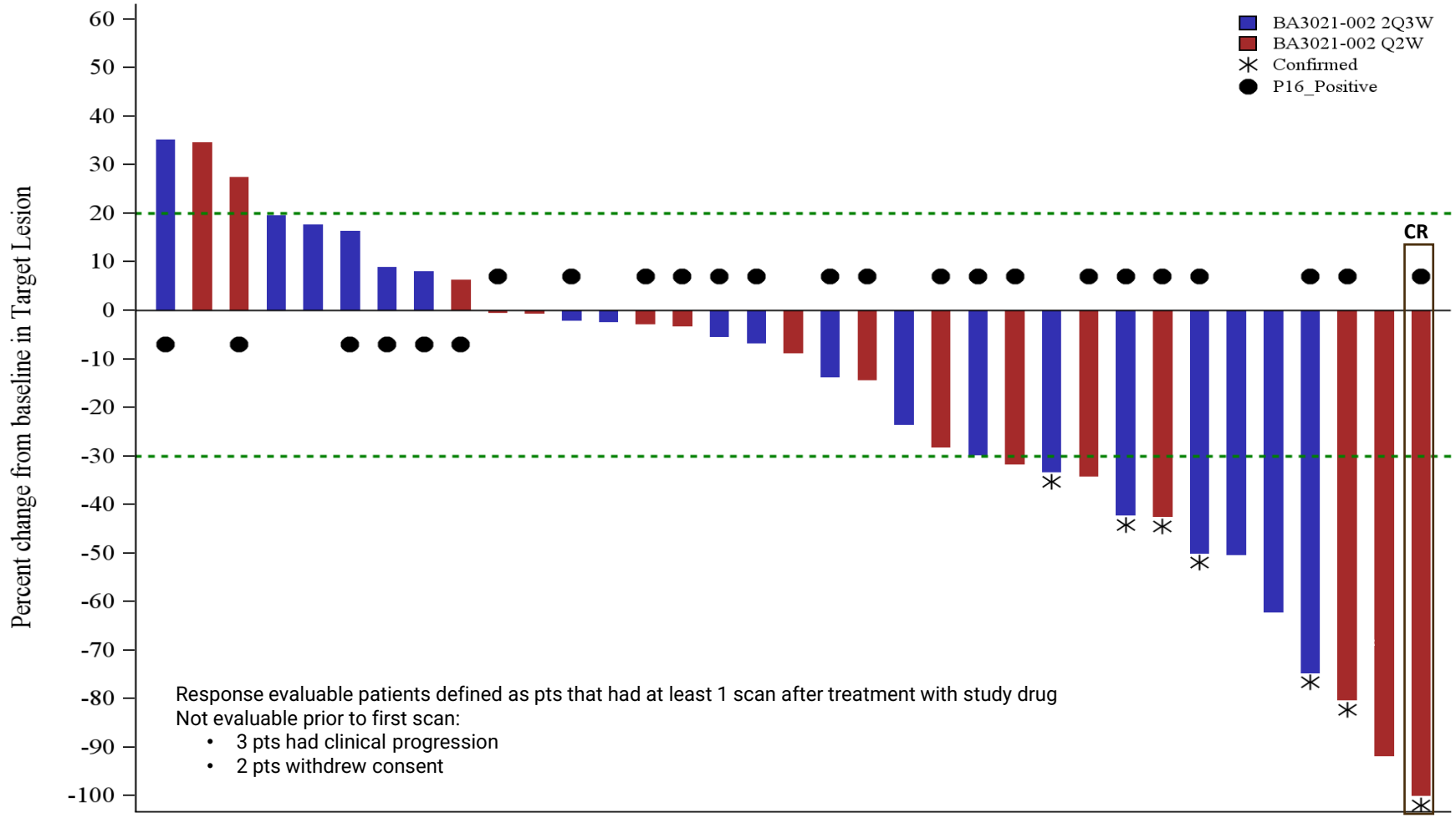
| Ozuriftamab vedotin 1.8 mg/kg | OPSCC p16+[a] | | Full Analysis |
|---|---|---|---|
| | 2Q3W (n=10) | Q2W (n=13) | 2Q3W and Q2W (N=40) |
| **Age, mean (SD), y** | 65 (5) | 62 (7) | 65 (8) |
| **Sex, n (%)** | | | |
| Male | 10 (100) | 12 (92) | 36 (90) |
| Female | 0 | 1 (8) | 4 (10) |
| **ECOG performance, n (%)** | | | |
| 0 | 3 (30) | 6 (46) | 15 (38) |
| 1 | 7 (70) | 7 (54) | 25 (63) |
| **Number of prior lines of therapy, median** | 3 | 3 | 3 |
| **Prior anti–PD-1 exposure, n (%)** | **10 (100)** | **13 (100)** | **40 (100)** |
| **Prior platinum-based chemotherapy exposure, n (%)** | **10 (100)** | **11 (85)** | **34 (85)** |
| **Prior taxane exposure, n(%)** | 7 (70) | 7 (54) | 26 (65) |

[a]HPV status was determined using p16 immunohistochemistry.
2Q3W = Days 1 and 8 of 21-day cycle

Data Cut Date: 31Oct2025

# Oz-V in SCCHN Continues to Demonstrate Clinical Responses in a Heavily Pretreated Population

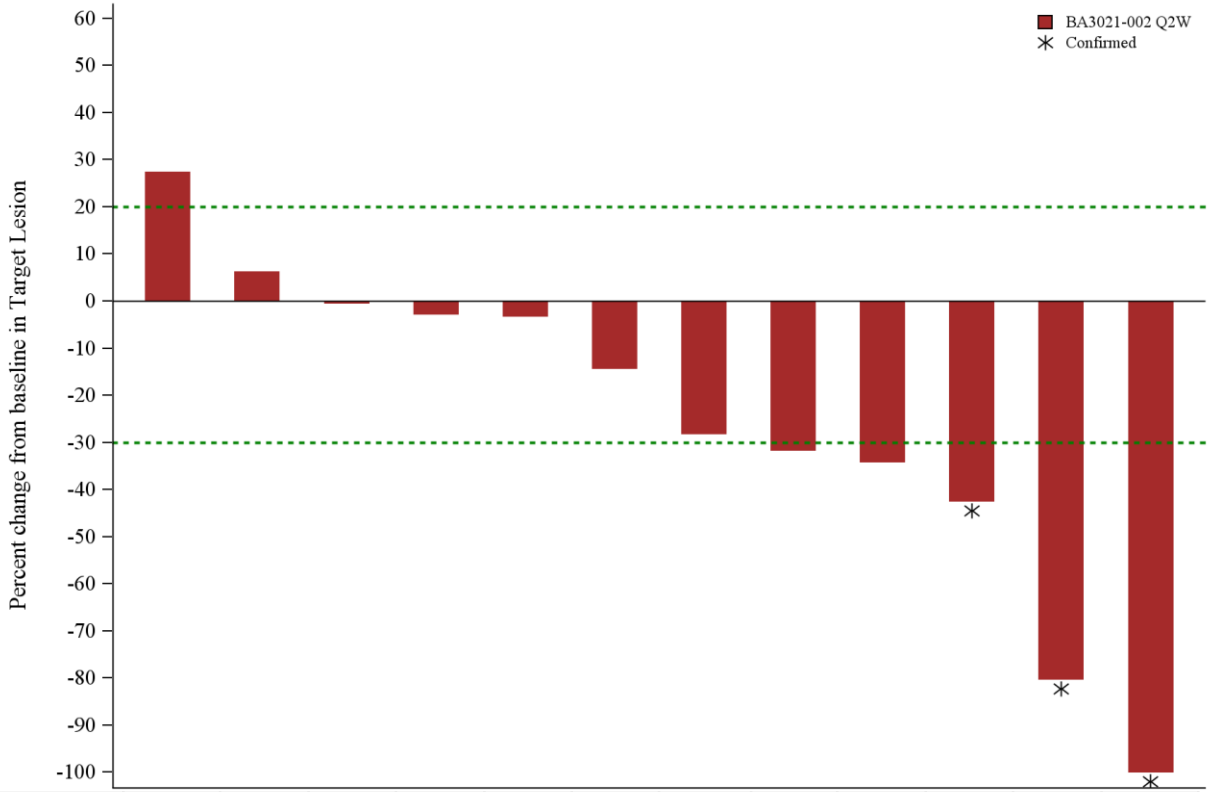
## 1.8 mg/kg Q2W and 2Q3W; Median of 3 prior lines of therapy




Data Cut Date: 31Oct2025

# Oz-V in SCCHN p16+^ OPSCC

## 1.8 mg/kg Q2W; Median of 3 prior lines of therapy




| Previous lines of therapy | 3 | 5 | 1 | 2 | 3 | 4 | 3 | 3 | 5 | 2 | 3 | 2 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|

| | Q2W |
|---|---|
| **Responders** (confirmed & unconfirmed) | **42%** (5/12*) |
| **Responders** (confirmed) | **25%** (3/12*) |
| **DCR** | **92%** (11/12*) |
| **Median DOR (months)** | ***9.9*** ***ongoing*** |
| **Median PFS (months)** | ***4.7*** ***ongoing*** |
| **Median OS (months)** | ***11.6*** ***ongoing*** |

^ p16 is strongly associated with HPV; HPV testing in progress for unknown patients

* Response evaluable patients defined as patients that had at least 1 scan after treatment with study drug

Not evaluable prior to first scan:

- 1 patients had clinical progression

Data Cut Date: 31Oct2025

# 2L+ HPV+ OPSCC: Cross Trial Comparisons of ORR and OS

## Considerably improved response rate and survival among a heavily pretreated trial population

| Cross trial comparisons** | Median prior lines of therapy | ORR (%) | OS (months)*** |
|---|---|---|---|
| **Ozuriftamab vedotin monotherapy (1.8 mg/kg Q2W)** *Study Ongoing** | **3** | **42% ORR 25% cORR** | **11.6 ongoing** |
| SOC (methotrexate, docetaxel, or cetuximab)[1,2] | 2 | 3.4% | 4.4 |
| Cetuximab monotherapy[3,4,5] | 1 | 0% | NA |
| Not approved | | | |
| Petosemtamab (1500 mg Q2W) | 2 | 13% | NA |

Accelerated Approval Opportunity (ORR)

Anti-EGFR therapies have inferior therapeutic outcomes among HPV+ patients

*Response evaluable patients defined as patients that had at least 1 scan after treatment with study drug;
Not evaluable prior to first scan: 1 patients had clinical progression

**The comparisons above are not based on data resulting from a head-to-head trial and are not direct comparisons. Different protocol designs, trial designs, patient selection and populations, number of patients, trial endpoints, trial objectives and other parameters that are not the same between the relevant trials may lead to bias in the results causing comparisons from different trials to be unreliable.

***From Dr. Alan Ho at MSKCC: "…extrapolating the OS of CheckMate 141 patients after progression on Nivo, we are figuring the OS of these patients to be about 7 months (OS of 9 -PFS of 2 months) post PD-1. Some of the retrospective papers (https://pubmed.ncbi.nlm.nih.gov/31574417/ & https://pubmed.ncbi.nlm.nih.gov/31864957/ we reviewed post-PD1 saw that upper limit of OS was of about 7-8.5 months post PD-1. **Figuring what the upper and lower estimates are, we think 6 months OS is a fair estimate for the null**."

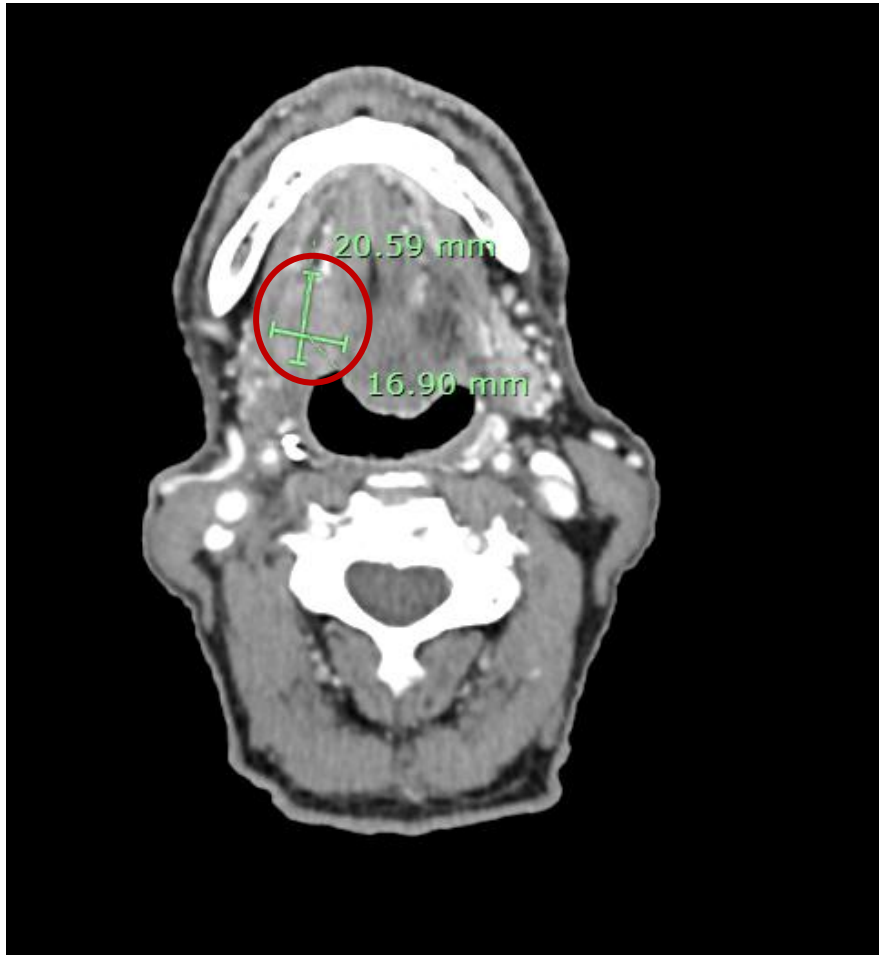
[1]N Engl J Med 2016;375:1856-1867. [2] Journal of Clinical Oncology 2018; 36(15): 1551-1558. [3]2008 Jun 15;112(12):2710-9. doi: 10.1002/cncr.23442, [4]Erbitux USPI accessed 2024., [5]INTERLINK-1: Phase 3 study of cetuximab ± monalizumab /Volume 34, Supplement 2S554-S555 October 2023
SOC, Standard of Care (Cetuximab, Methotrexate or Docetaxel); NA, not available

Data Cut Date: 31Oct2025

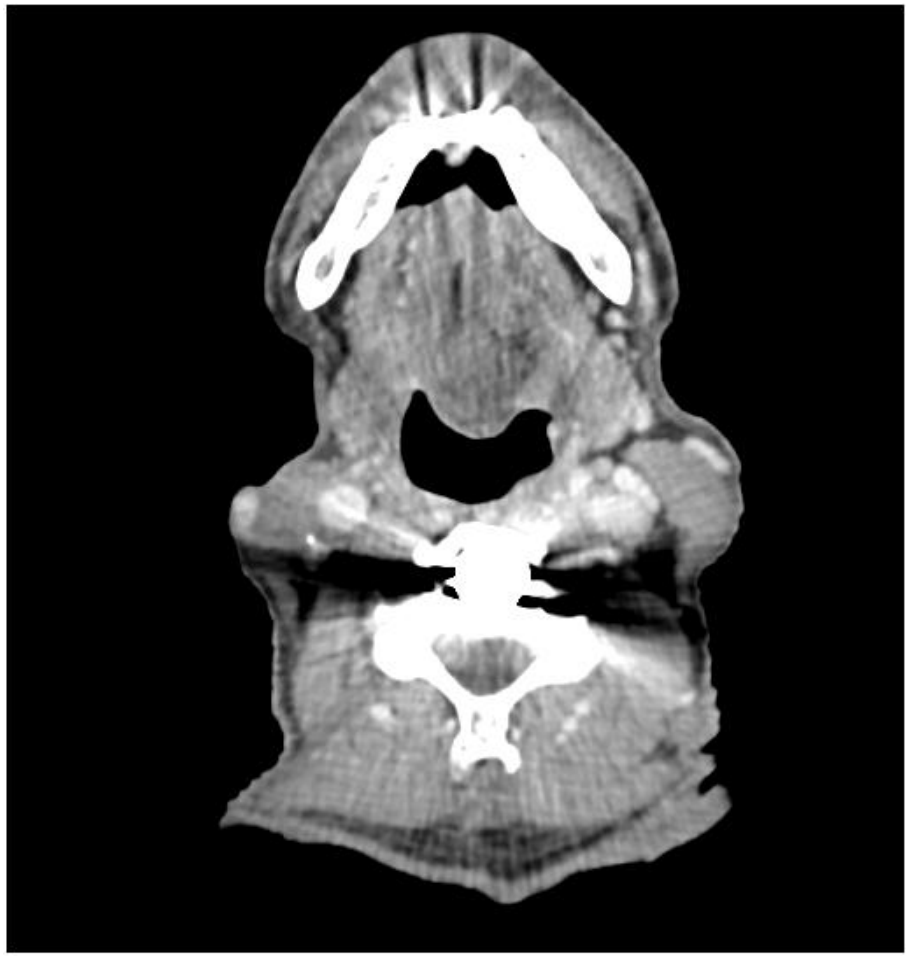
# Confirmed Complete Response Oz-V in SCCHN (1.8 mg/kg Q2W) – HPV Positive

**76-year-old male, stage IV – recurred after surgery and radiation therapy; prior treatments: pembrolizumab; clinical trial bispecific anti-PD1/CD47; patient remains in complete response >16 months after Oz-V treatment initiation**

Baseline - July 14, 2023



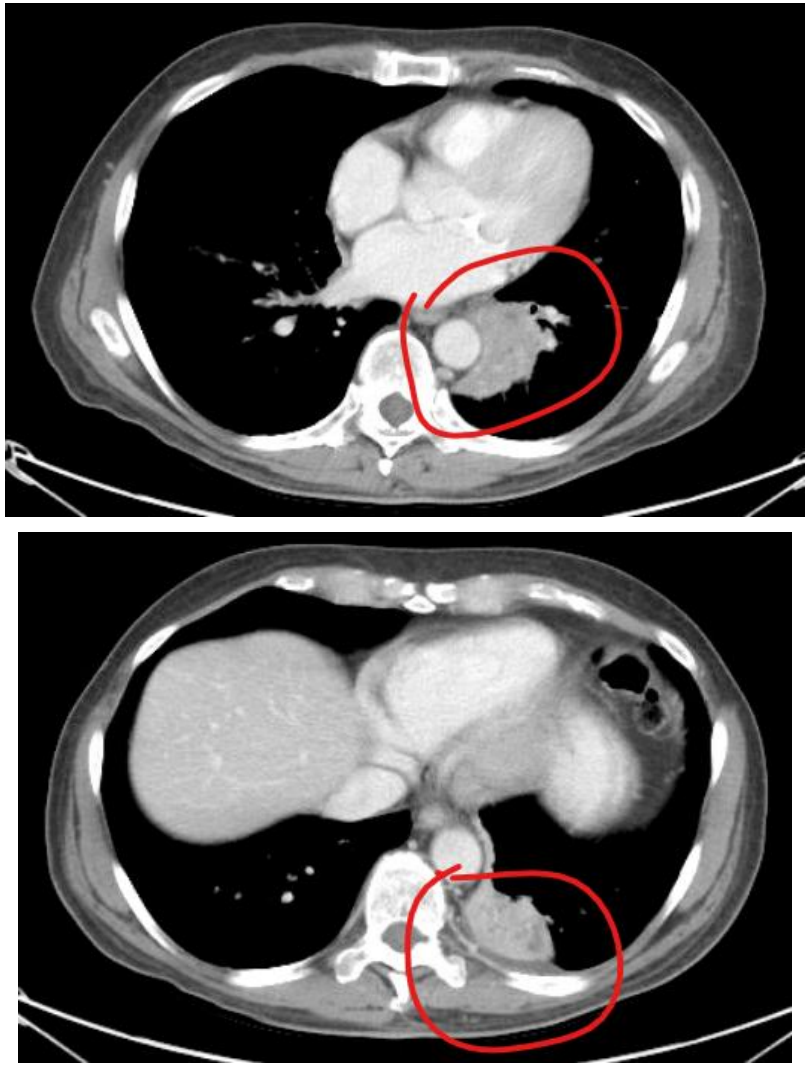

On Treatment – December 8, 2023



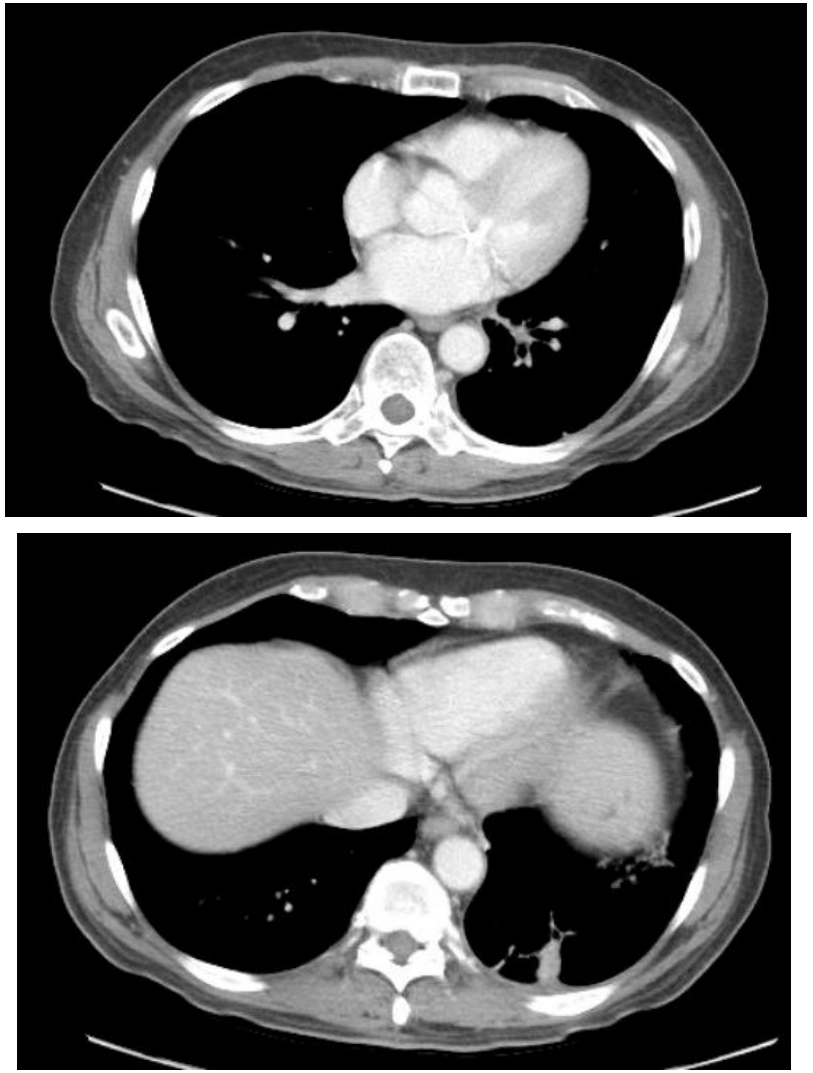
# Partial Response (-80%) Oz-V in SCCHN (1.8 mg/kg Q2W) – HPV Positive

## 63-year-old male, stage IV – recurred after surgery and radiation therapy; prior treatments: platinum, investigational agents including pembrolizumab

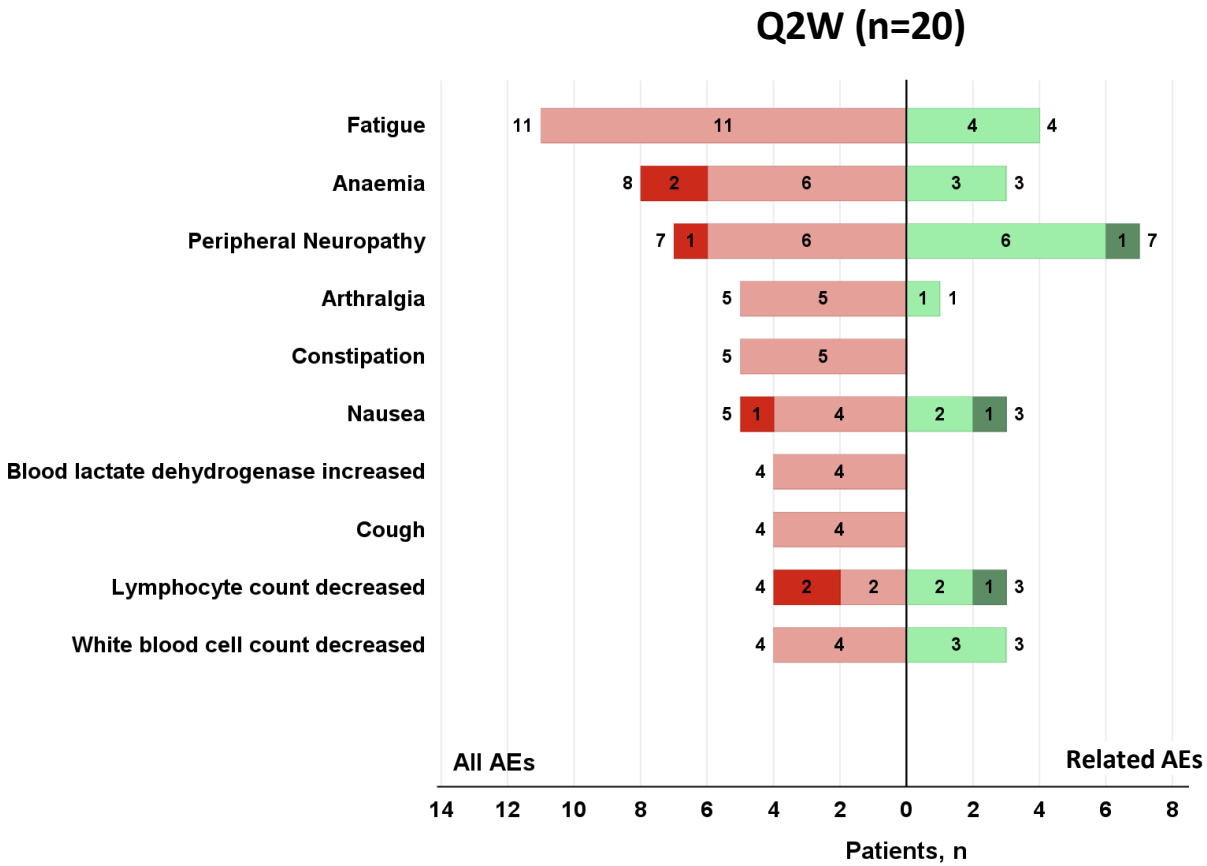
Baseline – December 4, 2025

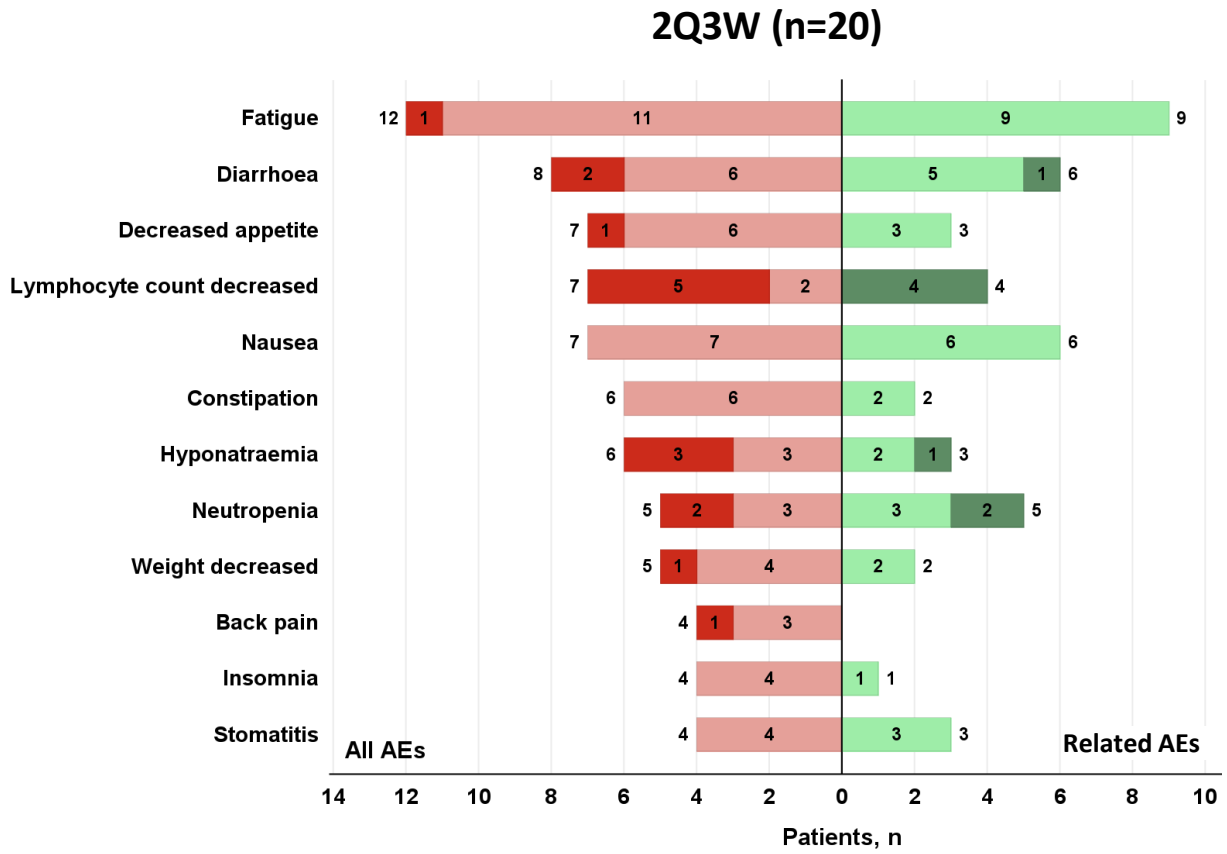


On Treatment – February 15, 2025

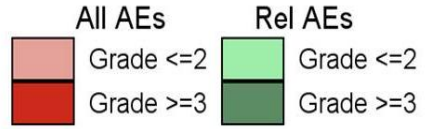


# Most frequent AEs of any grade (>15% of patients) (N=40)




| Treatment-Related Adverse Events | |
|---|---|
| Related Grade 3 | 3 (15%) |
| Related Grade 4 | 0 (0%) |
| Related Serious | 0 (0%) |




| Treatment-Related Adverse Events | |
|---|---|
| Related Grade 3 | 6 (30%) |
| Related Grade 4 | 2 (10%) |
| Related Serious | 2 (10%) |

Data Cut Date: 31Oct2025

# Ph2 Oz-V: Overall Safety Summary of SCCHN patients

## Investigator Choice[3] related AEs G3 or 4 = 35% - Considerably higher than Oz-V 1.8 mg/kg Q2W

| | 1.8 mg/kg Q2W (N=20) | 1.8 mg/kg 2Q3W (N=20)[4] | Total (N=40)[4] |
|---|---|---|---|
| Any Adverse Events (AEs) | 19 (95%) | 20 (100%) | 38 (95%) |
| Related AEs with CTCAE[1] Grade 3 or 4[2] | **3 (15%)** | 7 (35%) | 10 (25%) |
| Any related serious AEs[2] | 0 | 2 (10%) | 2 (5%) |
| Possibly Related AEs leading to death[2] | 0 | 0 | 0 |
| Related AEs leading to treatment discontinuation[2] | 1 (5%) | 1 (5%) | 2 (5%) |

[1]CTCAE: Common Terminology Criteria for Adverse Events. The NCI Common Terminology Criteria for Adverse Events is a descriptive terminology which is utilized for Adverse Event (AE) reporting. A grading (severity) scale is provided for each AE term.
[2]As assessed by the investigator. Missing responses are counted as related.
[3]N Engl J Med 2016;375:1856-1867. [2] Journal of Clinical Oncology 2018; 36(15): 1551-1558
[4]One patient from Phase 1 not included

Data Cut Date: 31Oct2025

# Phase 2 Oz-V Safety Data

## Most frequent treatment-emergent related adverse events (>15%)

| | 1.8 mg/kg Q2W (N=20) | | 1.8 mg/kg 2Q3W (N=20)^ | | Total (N=40)^ | |
|---|---|---|---|---|---|---|
| **Preferred Term** | **All Grades n (%)** | **Grade 3-4 n (%)** | **All Grades n (%)** | **Grade 3-4 n (%)** | **All Grades n (%)** | **Grade 3-4 n (%)** |
| **Number of subjects with at least one TRAE** | 19 (95) | 12 (60) | 20 (100) | 16 (80) | 39 (98) | 28 (70) |
| Fatigue | 11 (55) | 0 | 12 (60) | 1 (5) | 23 (58) | 1 (3) |
| Nausea | 5 (25) | 1 (5) | 7 (35) | 0 | 12 (30) | 1 (3) |
| Anaemia | 8 (40) | 2 (10) | 3 (15) | 1 (5) | 11 (28) | 3 (8) |
| Constipation | 5 (25) | 0 | 6 (30) | 0 | 11 (28) | 0 |
| Lymphocyte count decreased | 4 (20) | 2 (10) | 7 (35) | 5 (25) | 11 (28) | 7 (18) |
| Decreased appetite | 3 (15) | 0 | 7 (35) | 0 | 10 (25) | 1 (3) |
| Peripheral neuropathy¥ | 7 (35) | 1 (5) | 3 (15) | 1 (5) | 10 (25) | 2 (5) |
| Diarrhoea | 1 (5) | 0 | 8 (40) | 2 (10) | 9 (23) | 2 (5) |
| Hyponatraemia | 3 (15) | 1 (5) | 6 (30) | 4 (20) | 9 (23) | 5 (13) |
| Arthralgia | 5 (25) | 0 | 3 (15) | 0 | 8 (20) | 0 |
| Weight decreased | 3 (15) | 0 | 5 (25) | 1 (5) | 8 (20) | 1 (3) |
| Cough | 4 (20) | 0 | 3 (15) | 0 | 7 (18) | 0 |
| Blood lactate dehydrogenase increased | 4 (20) | 0 | 3 (15) | 0 | 7 (18) | 0 |
| Hypercalcaemia | 3 (15) | 1 (5) | 3 (15) | 1 (5) | 6 (15) | 2 (5) |
| Neutropenia* | 1 (5) | 0 | 5 (25) | 2 (10) | 6 (15) | 2 (5) |
| White blood cell count decreased | 4 (20) | 0 | 2 (10) | 0 | 6 (15) | 0 |

^ One patient from Phase 1 not included

* Derived from neutropenia, and neutrophil count decreased

¥ Derived from neuropathy peripheral, peripheral motor neuropathy, and peripheral sensory neuropathy

Data Cut Date: 31Oct2025

# FDA End of Phase 2 Meeting: Key Outcomes

## Dual primary endpoints of ORR and OS to support potential accelerated approval followed by full approval

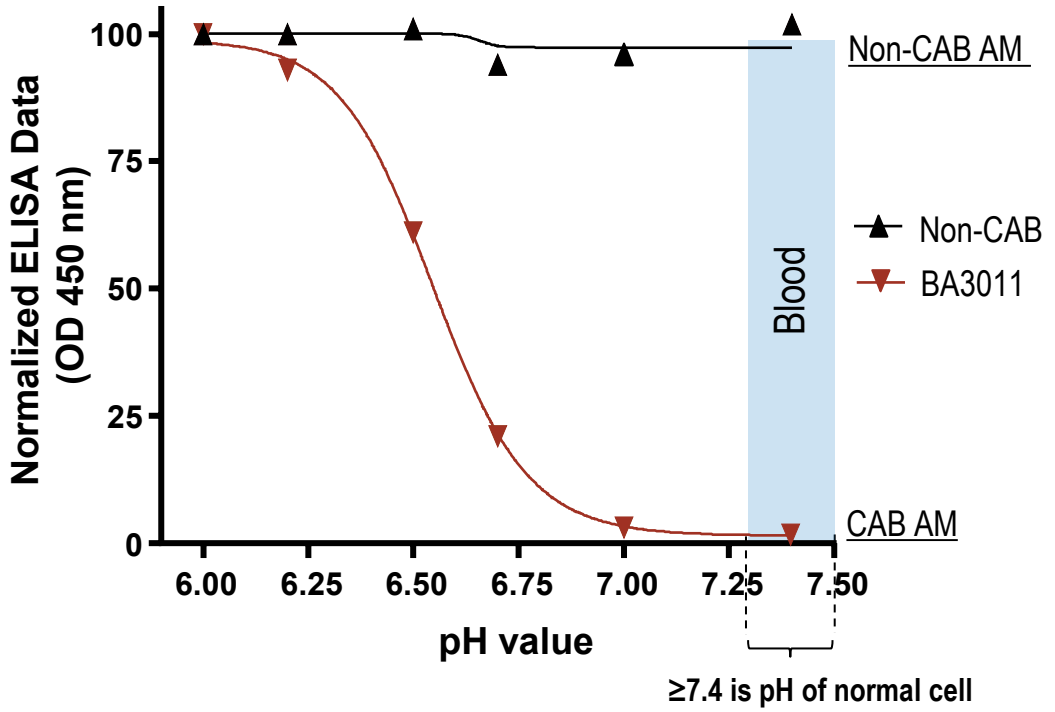
- **Pivotal Trial Design:** For full approval, approximately 300 patients prospectively randomized and stratified, one to one between two open label treatment arms
- **Oz-V Dose and Regimen:** Patients randomized to the investigational arm will receive 1.8 mg/kg every other week
- **Investigator's Choice (IC) control arm:** Patients randomized to the control arm will receive either cetuximab, docetaxel, or methotrexate monotherapy
- **Accelerated Approval Endpoint:** Based on interim analysis of enrolled patients, statistically significant improvement of confirmed Overall Response Rate (ORR) by Blinded Independent Central Review (BICR) supported by an adequately characterized Duration of Response (DOR) without detriment in OS
- **Full Approval Endpoint:** Statistically significant improvement of OS

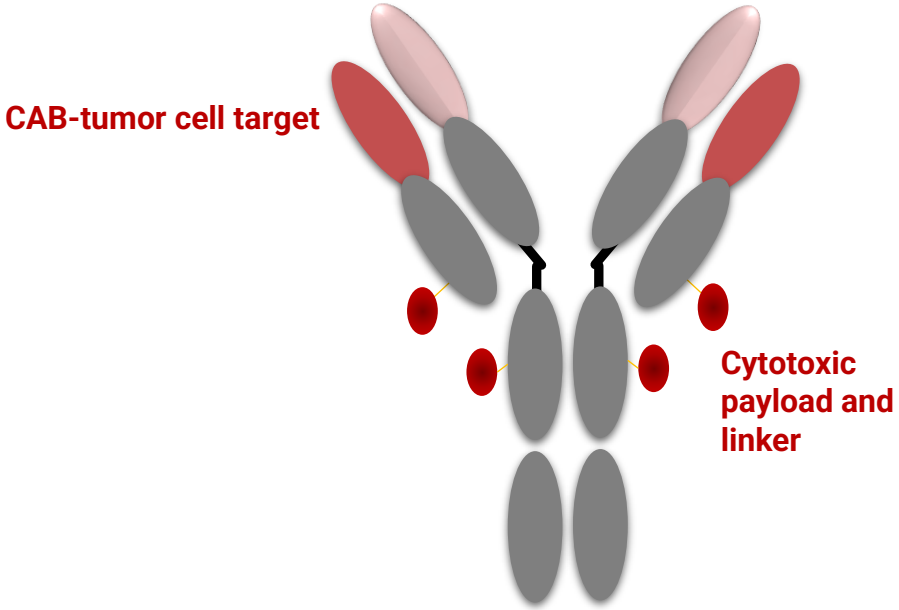
# Mecbotamab Vedotin (CAB-AXL-ADC): mKRAS Non-Small Cell Lung Cancer (NSCLC)

# Mecbotamab Vedotin (Mec-V): CAB-AXL-ADC

## AXL is expressed in a variety of tumor types, with overexpression associated with metastasis, tumor resistance to chemotherapy, and poor prognosis

**BA3011 pH binding inflection point adjusted for tumor microenvironment selectivity**



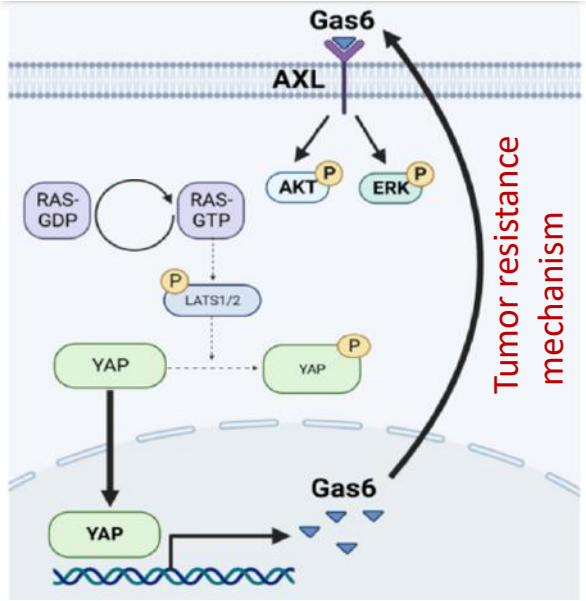

- Humanized anti-AXL IgG1
- ~100 pM affinity (pH 6.5)
- VC-MMAE (DAR 4) linker and payload
- Epitope in Ig loop region

1. Gay CM, Balaji K, Byers LA. *Br J Cancer*. 2017;116(4):415-423. 2. Zhang G, Wang M, Zhao H, Cui W. *Oncol Lett*. 2018;15(3):2726-2734.
Abbreviations: ADC, antibody-drug conjugate; AM, affinity matched; CAB, conditionally active biologic; DAR, drug antibody ratio; ELISA, enzyme linked immunosorbent assay; Ig, immunoglobulin; OD, optical density; VC-MMAE, valine-citrulline monomethylauristatin E.

# AXL Plays a Crucial Role in the Survival of mutated KRAS (mKRAS) NSCLC Cells

- mKRAS represents 30% of all NSCLC patients
- 70% to 85% of mKRAS NSCLC express AXL by IHC and higher by mRNA analysis
- AXL over-expression drives aggressive tumor characteristics, resistance to therapies, and poor patient outcomes
- Significant opportunity for Mec-V (CAB-AXL-ADC)

**mKRAS leads to upregulation and activation of AXL expression**

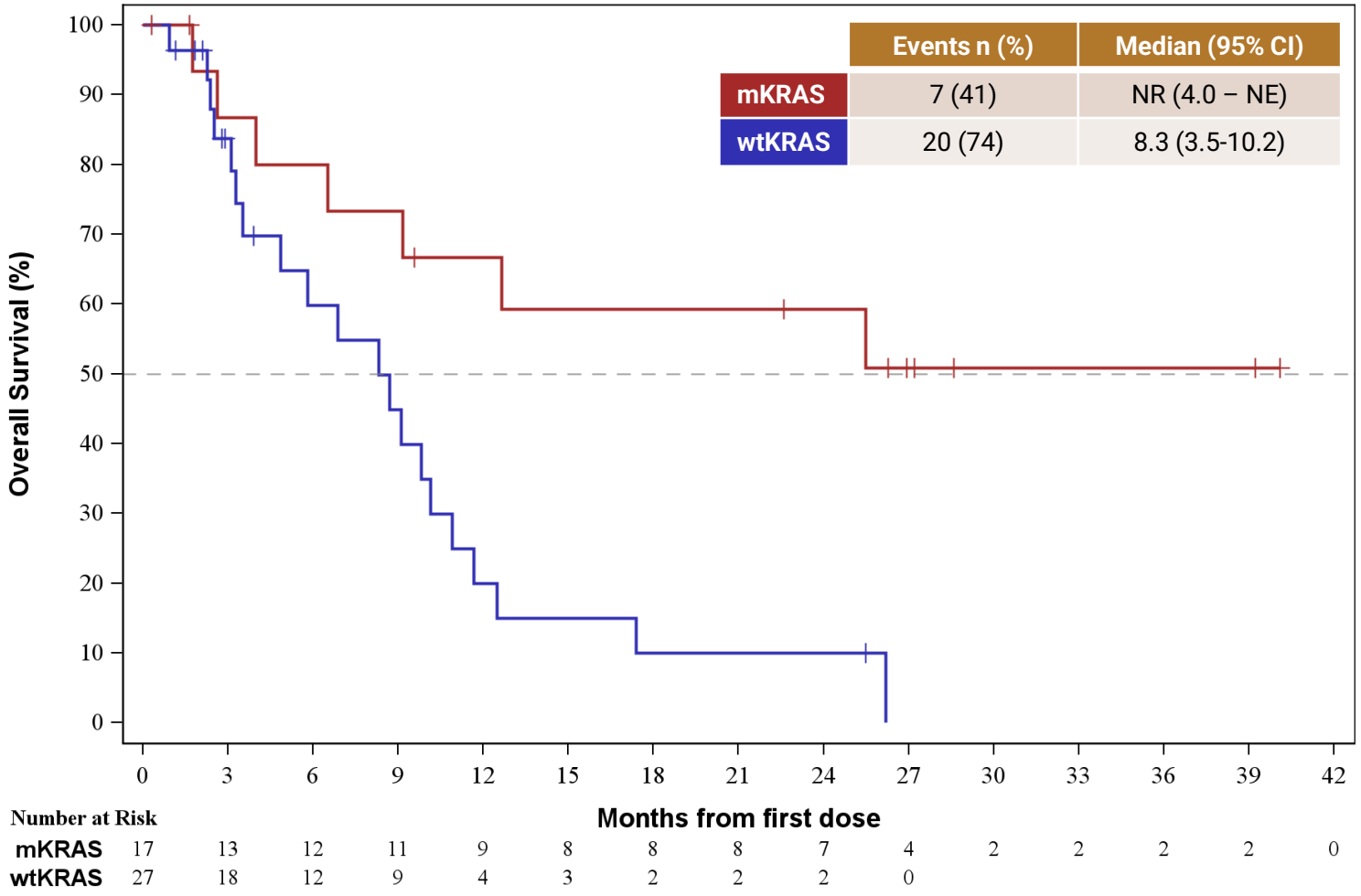



***Adapted from Morimoto et al. Cancer Letters 587 (April 2024)***

* Morimoto et al., Cancer Letters 587 (2024) 216692 and BioAtla study BA3011-002
IHC- Immunohistochemical assay

# Mec-V 1.8 mg/kg Q2W Overall Survival mKRAS vs wtKRAS NSCLC

## Median of 3 prior lines of tx




| median of 2 prior lines of tx | mKRAS (Q2W only) |
|---|---|
| Responders (confirmed & unconfirmed) | 31% (5/16^) |
| Responders (confirmed) | 19% (3/16^) |
| DCR | 81% (13/16^) |
| PFS | 4.6 |
| One-year landmark OS | 67% |

^Response evaluable patients defined as patients that had at least 1 scan after treatment with study drug
Prior to first scan: one patients withdrew consent

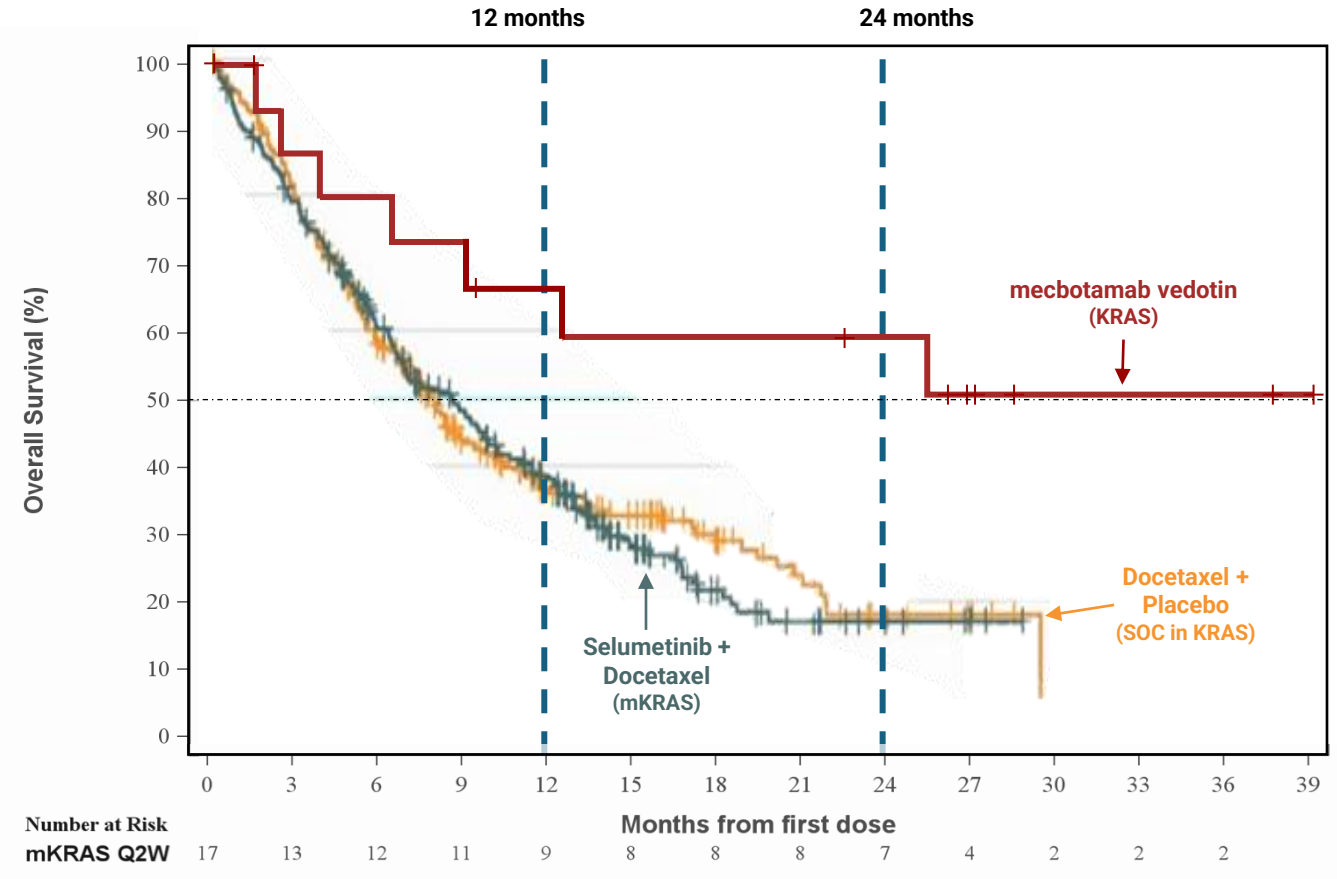
Data Cut Date: 19Aug2025

# Mec-V 1.8 mg/kg Q2W Associated with Exceptional Overall Survival in mKRAS NSCLC

## Overall survival cross trial comparison[1]

| | mecbotamab vedotin (1.8 mg/kg Q2W) | Docetaxel[3] |
|---|---|---|
| **Population** | mKRAS | mKRAS |
| **Number of patients** | 17 | 256 |
| **Prior Lines of tx** | 2 | 1 |
| **Prior Taxane** | **53%** | **0%** |
| **ORR** | 31%[2] | 14% |
| **PFS (months)** | 4.5 | 2.8 |
| **OS (months)** | Not reached | 7.9 |
| **Survival at 12 months** | 67% | <40% |
| **Survival at 24 months** | 59% | <20% |

***FDA guidance: 2L+ NSCLC Phase 3 trial will be randomized mecbotamab vedotin versus docetaxel (full-approval = OS)***




[1] The comparisons above are not based on data resulting from a head-to-head trial and are not direct comparisons. Different protocol designs, trial designs, patient selection and populations, number of patients, trial endpoints, trial objectives and other parameters that are not the same between the relevant trials may lead to bias in the results causing comparisons from different trials to be unreliable.

[2] Confirmed and unconfirmed responders.

[3] JAMA. 2017 May 9;317(18):1844–1853

Data Cut Date: 19Aug2025

# Competitive Response Rate in 2L+ mKRAS NSCLC

## Median Overall Survival ranges from 6 to 11 months in mKRAS NSCLC when treated with docetaxel in the 2L+

| Cross trial comparisons** | Pts | Median prior lines of therapy | ORR | Median OS (months) |
|---|---|---|---|---|
| **Mecbotamab Vedotin (1.8 mg/kg Q2W regimen)** *Study Ongoing** | **17** | **3** | **31%*** ** | **Not Reached (1 year landmark at 67%)** |
| SELECT-1[1] (all mKRAS variants) | 256 | 1 | 13.7% | 7.9 |
| Real-life ESME cohort[2] (all mKRAS variants) | 1000+ | 1 | NA | 6.1 to 10.6* |
| Codebreak 200[3] **(mKRAS G12C)** | 174 | 2 | 13.2% | 11.3 |
| KRYSTAL-12[4] **(mKRAS G12C)** | 152 | 2 | 9.2% | NA |

Docetaxel Studies

1. Janne, P et al. JAMA. 2017 May 9;317(18):1844–1853. (2) Thomas QD, et al. ESMO Open, Volume 9, Issue 6, 103473. (3) de Langen AJ, et al. Lancet 2023; 401:733-746; (4) Mok TS, Journal Clinical Oncology 2024; 42(17_suppl):LBA8509.

* PD-1/PD-L1 monotherapy, PT-based CT without a PD-1/PD-L1, or Docetaxel monotherapy or combination

**The comparisons above are not based on data resulting from a head-to-head trial and are not direct comparisons. Different protocol designs, trial designs, patient selection and populations, number of patients, trial endpoints, trial objectives and other parameters that are not the same between the relevant trials may lead to bias in the results causing comparisons from different trials to be unreliable.

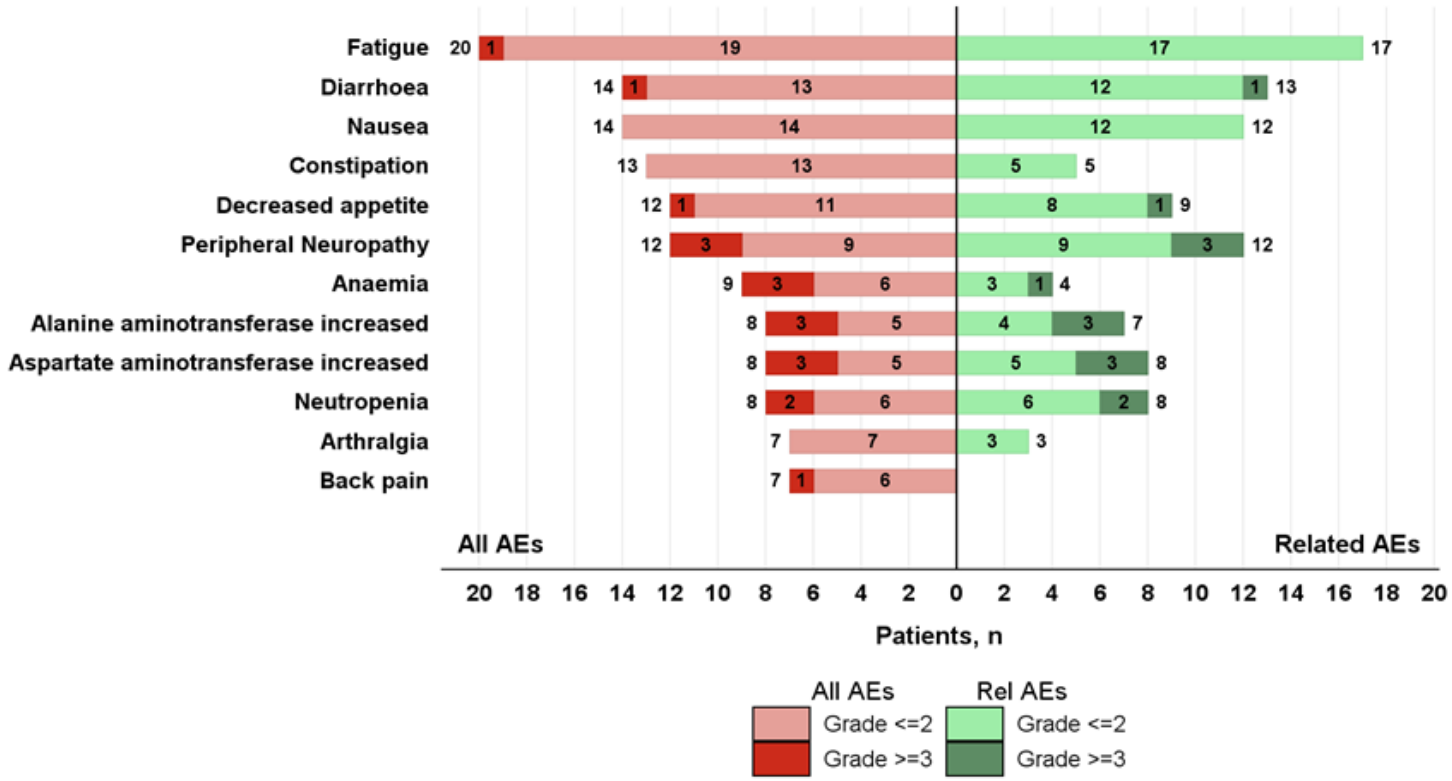
***Confirmed and Unconfirmed

# Ph2 Mec-V: Overall Safety Summary of NSCLC patients

## 1.8 mg/kg Q2W with or without nivolumab generally well-tolerated; only 7% discontinuation due to related AEs

| | N=45 (%) |
|---|---|
| Any Adverse Events (AEs) | 45 (100) |
| Related AEs with CTCAE[1] Grade 3 or 4[2] | 14 (31) |
| Any Related Serious AEs[2] | 6 (13) |
| Possibly Related AEs leading to death[2] | 0 |
| Related AEs leading to treatment discontinuation[2] | 3 (7) |

### Most frequent AEs any grade occurring at a rate >15%




[1]CTCAE: Common Terminology Criteria for Adverse Events. The NCI Common Terminology Criteria for Adverse Events is a descriptive terminology which is utilized for Adverse Event (AE) reporting. A grading (severity) scale is provided for each AE term.
[2]As assessed by the investigator. Missing responses are counted as related.

Data Cut Date: 19Aug2025

# Potential for Mec-V to Address All mKRAS NSCLC

**1.8 mg/kg Q2W associated with improved overall survival and favorable benefit / risk profile**

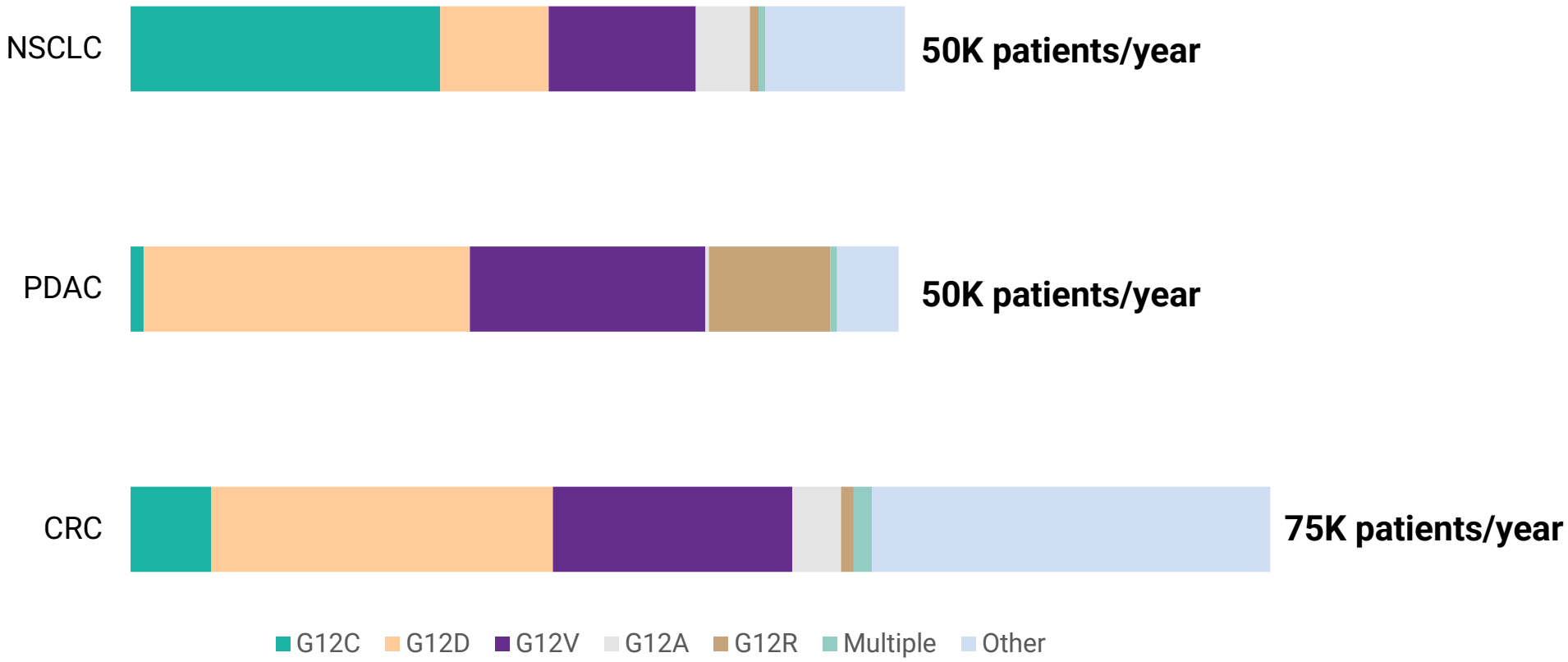
- Promising anti-tumor activity among patients whose tumors express KRAS mutations
  - mKRAS represents 30% of all NSCLC patients and is associated with increased AXL expression
  - 1.8 mg/kg Q2W associated with exceptional overall survival even in heavily pretreated patients
  - 67% alive at a landmark of one year
  - 59% alive at a landmark of two years; standard of care agents result in less than 20% alive at the two year*
  - Anti-tumor activity across nine different KRAS mutation variants
    - Partial response observed in a patient who had experienced prior failure of sotorasib
    - Patient treated with mecbotamab vedotin + anti-PD-1 antibody remains in complete response for >2 years
- Potential for a pan mKRAS strategy in NSCLC; currently positioning for a future pivotal trial

*The comparisons above are not based on data resulting from a head-to-head trial and are not direct comparisons. Different protocol designs, trial designs, patient selection and populations, number of patients, trial endpoints, trial objectives and other parameters that are not the same between the relevant trials may lead to bias in the results causing comparisons from different trials to be unreliable.

# Significant Opportunity for Mec-V to Expand Beyond mKRAS NSCLC

## KRAS mutations most commonly found in CRC, NSCLC and PDAC

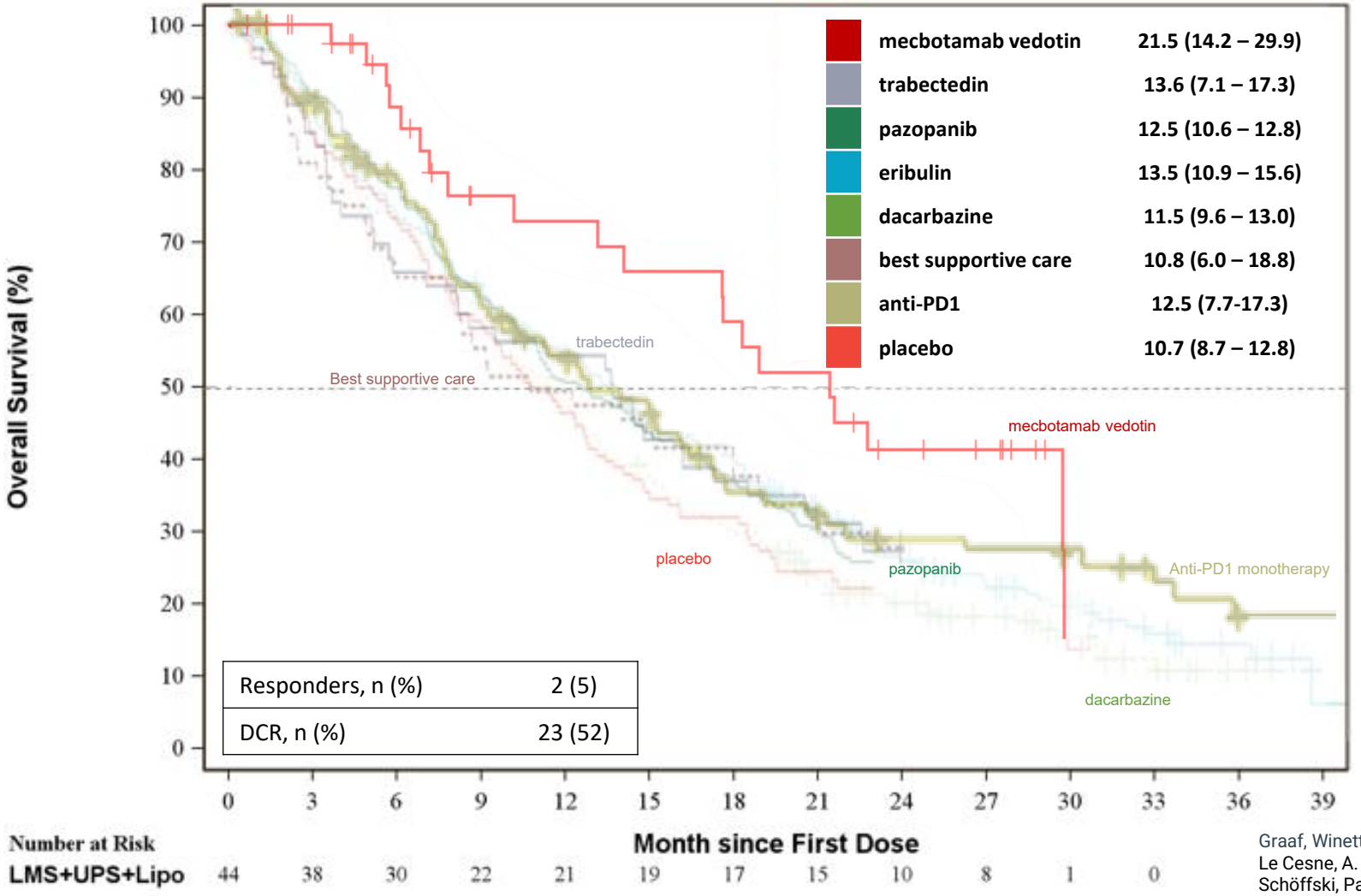
Estimated US incidence of select mKRAS cancers and distribution of selected mKRAS variants




Lee J., Sivakumar S., Schrock A., et al. NPJ Precision Oncology, 2022. PMID: 36494601.
CRC: colorectal cancer; NSCLC: non-small cell lung cancer; PDAC: pancreatic ductal adenocarcinoma

# Improved Overall Survival also Observed in Soft Tissue Sarcoma Mirrors Overall Survival Observed in mKRAS NSCLC

## LMS, UPS, and Liposarcoma: 1.8 mg/kg Q2W Mono and anti-PD-1 Combo; cross-trial comparison*




Data Cut Date: 24Mar2025

## Mec-V in Sarcoma:

**Median of 2 prior lines of Tx**

**Median OS of 21.5 months**

**Landmark OS at 1 year 73% (54-85)**

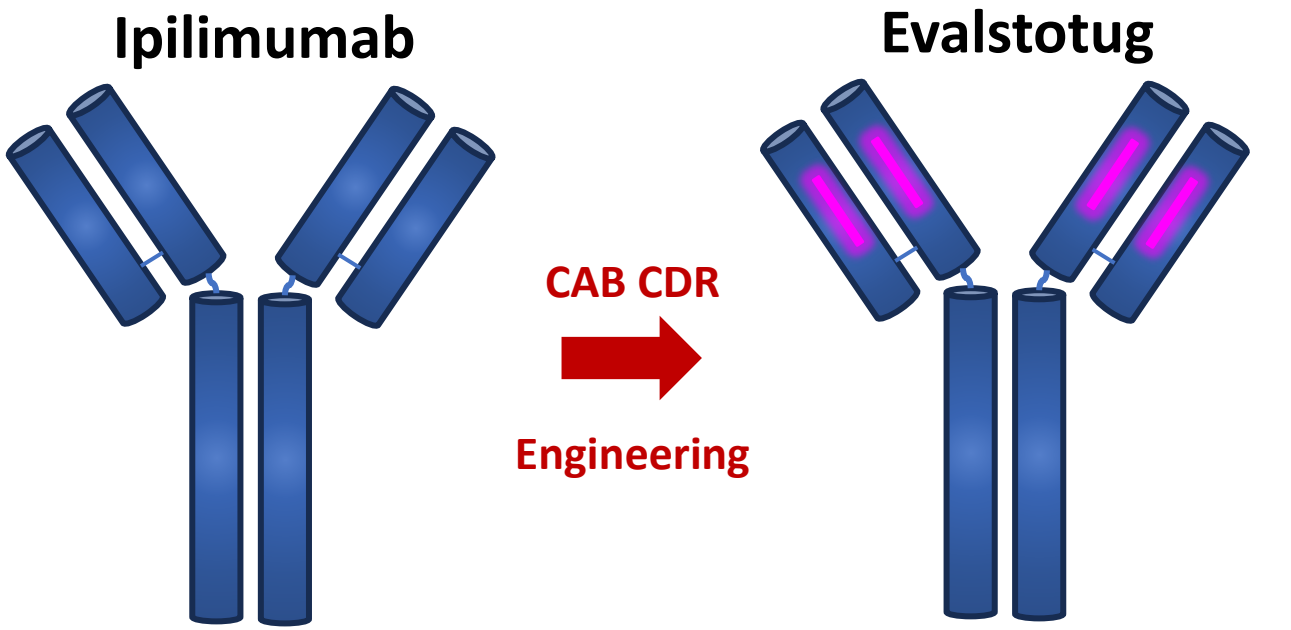

Graaf, Winette van der et al. *The Lancet* 379 (2012): 1879-1886.
Le Cesne, A. et al Annals of Oncology, Volume 32, Issue 8, 1034 - 1044
Schöffski, Patrick et al. The Lancet, Volume 387, Issue 10028, 1629 – 1637
Miao, R. *Cancer Immunol Immunother* 72, 2521–2527 (2023)


*The comparisons above are not based on data resulting from a head-to-head trial and are not direct comparisons. Different protocol designs, trial designs, patient selection and populations, number of patients, trial endpoints, trial objectives and other parameters that are not the same between the relevant trials may lead to bias in the results causing comparisons from different trials unreliable.

# Evalstotug (CAB-CTLA-4)

# Evalustotug is a Next Generation Adaptation of Ipilimumab

## CAB-CTLA4 selectively active in tumor microenvironment, thereby reducing immune mediated adverse events (imAEs)




Inability to distinguish between normal and tumor tissue

Similar efficacy and PK enhanced with selective pH-dependent tumor binding

# Evalstotug is a “CABified” Ipilimumab: A Next Generation CTLA-4 Inhibitor

## Preserved efficacy with reduced toxicity in combination with PD-1

- Ipilimumab (ipi) CDRs modified to bind at tumor cell acidic pH, but not at normal pH leading to evalstotug:
  - Preserved affinity and epitope
  - Equivalent $E_{Max}$ and $EC_{50}$ in preclinical models
  - However, observed substantially reduced G.I. toxicity in primates

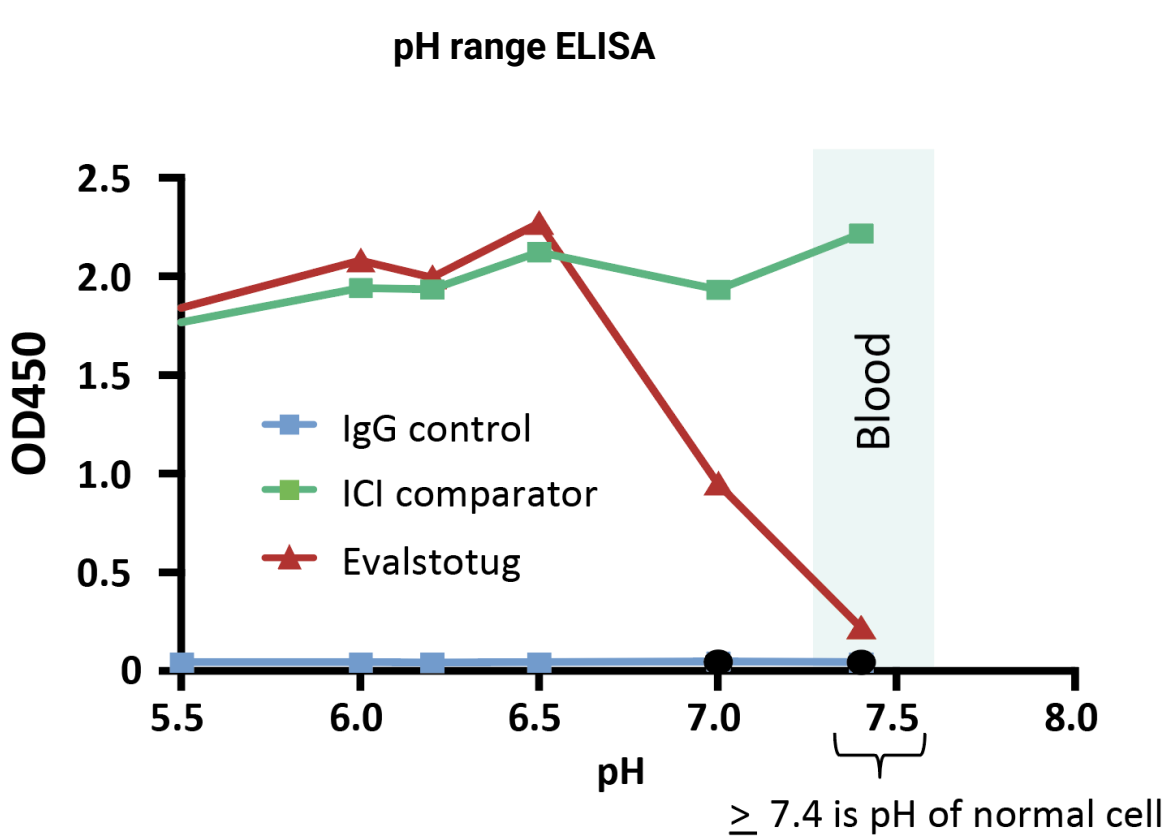
- CAB CTLA-4, evalstotug, enables targeted exposure in TME enabling lower imAE relative to ipi

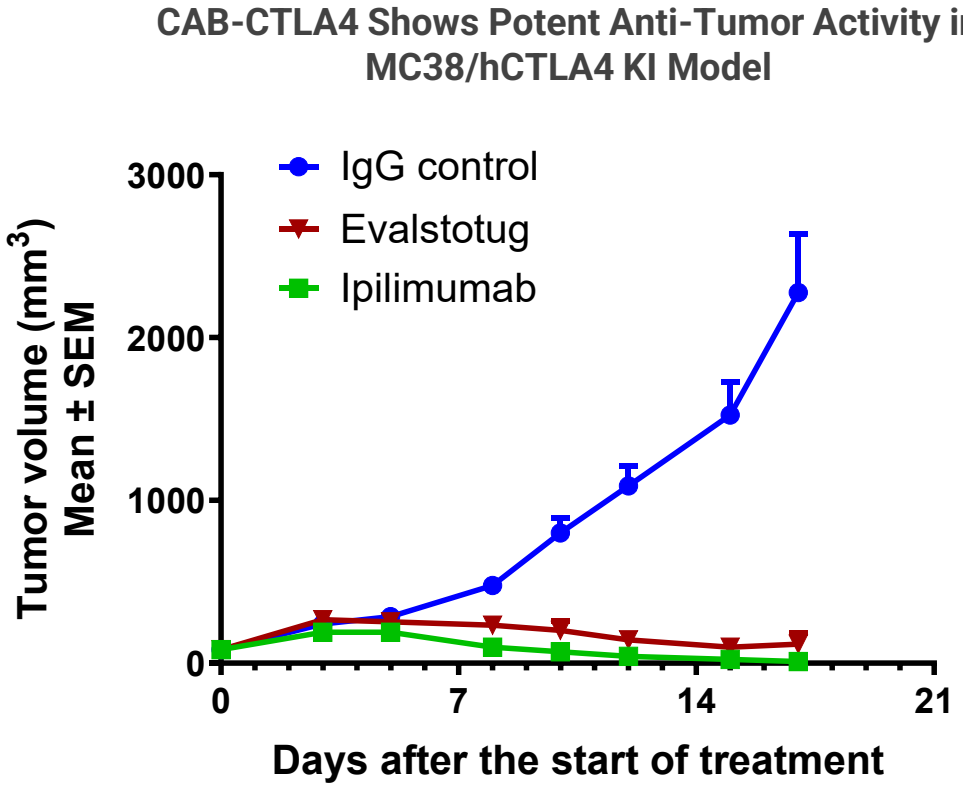
Abbreviations: ADCC, antibody-dependent cellular cytotoxicity; CAB, Conditionally Active Biologic; CD, cluster of differentiation; CDR, complementarity-determining region; CTLA-4, cytotoxic T-lymphocyte associated protein 4; EC50, concentration producing 50% Emax; Emax, maximum effect; imAE, immune mediated adverse event; PD-1, programmed cell death protein 1; t1/2, half-life; Treg, regulatory T cells.

1. Chang HW, et al. Proc Natl Acad Sci USA. 2021;118(9):e2020606118.

# Reversible Binding in the TME

## Comparison of evalstotug binding to CTLA-4 in different pH conditions



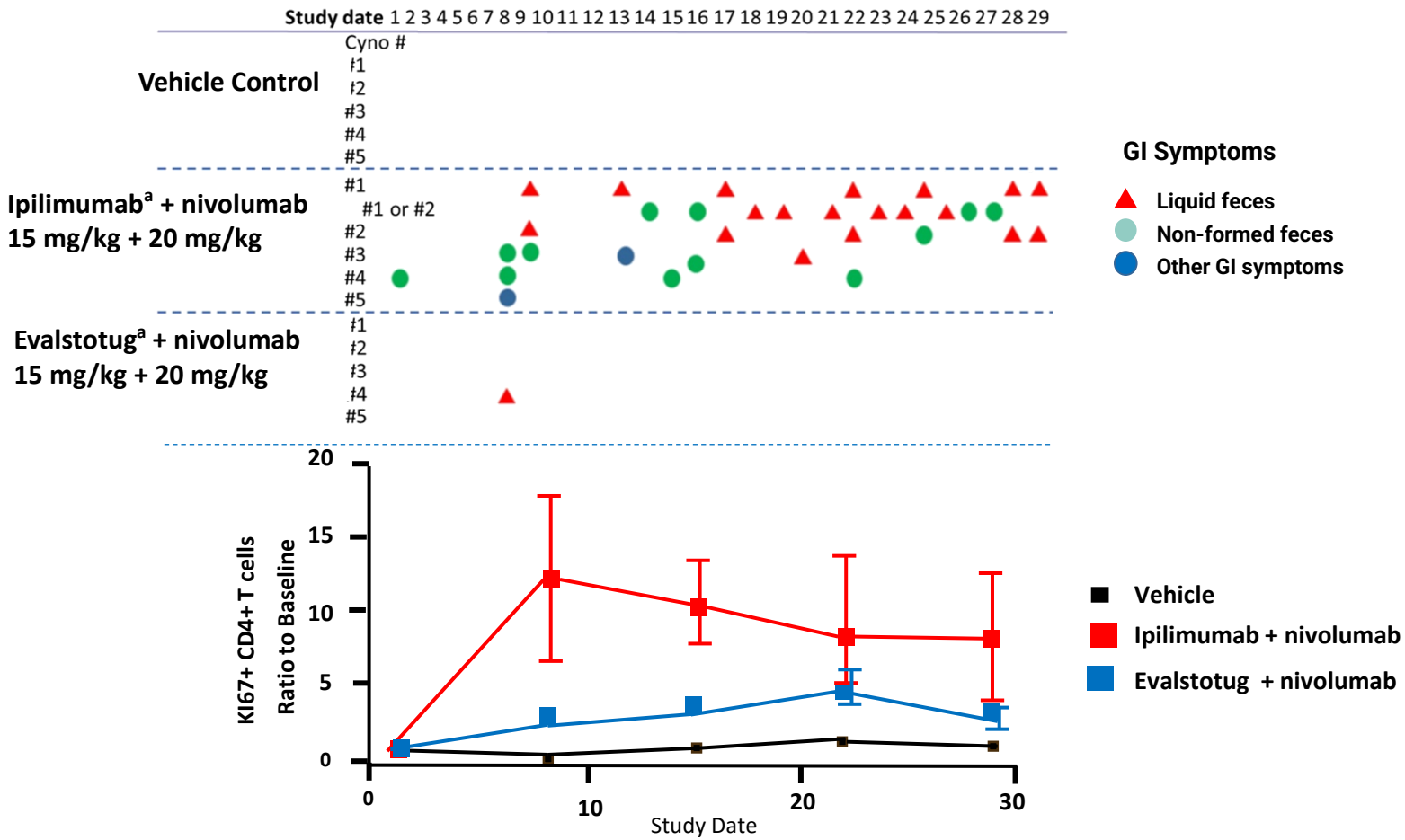




**BA3071 induces complete tumor regression in mouse tumor model**. Mice were dosed with IgG control and anti-CTLA antibodies at 10mg/kg (equivalent to 1mg/kg anti-CTLA-4 human dose), IP, BIW, N=12 mice/group.

Abbreviations: CTLA-4, cytotoxic T-lymphocyte associated protein 4; ICI, immune checkpoint inhibitor; IgG, immunoglobulin G; OD450, optical density at 450 nm; TME, tumor microenvironment.

Note: Figures modified from Chang HW, et al. Proc Natl Acad Sci USA. 2021;118(9):e2020606118.

# Evalstotug Reduced GI Toxicity in Primates




Abbreviations: CD, cluster of differentiation; Cyno, cynomolgus macaque; GI, gastrointestinal; QW, once weekly.
Note: Ipilimumab and evalstotug had the same half-life and exposure in this model. Figure modified from Chang HW, et al. Proc Natl Acad Sci USA. 2021;118(9):e2020606118.
[a]Ipilimumab analog or evalstotug 15 mg/kg (≈11 mg/kg human dose) + nivolumab 20 mg/kg (≈14.6 mg/kg human dose) both administered QW for 4 weeks.

# Evalstotug Across Multiple Tumor Types

# Evalstotug at 350mg Q3W (5mg/kg based on 70 kg pt) in Combination With PD1: Demographics – Across Multiple Tumor Types; n=17

## 82% patients experienced failure of prior PD-1 treatment

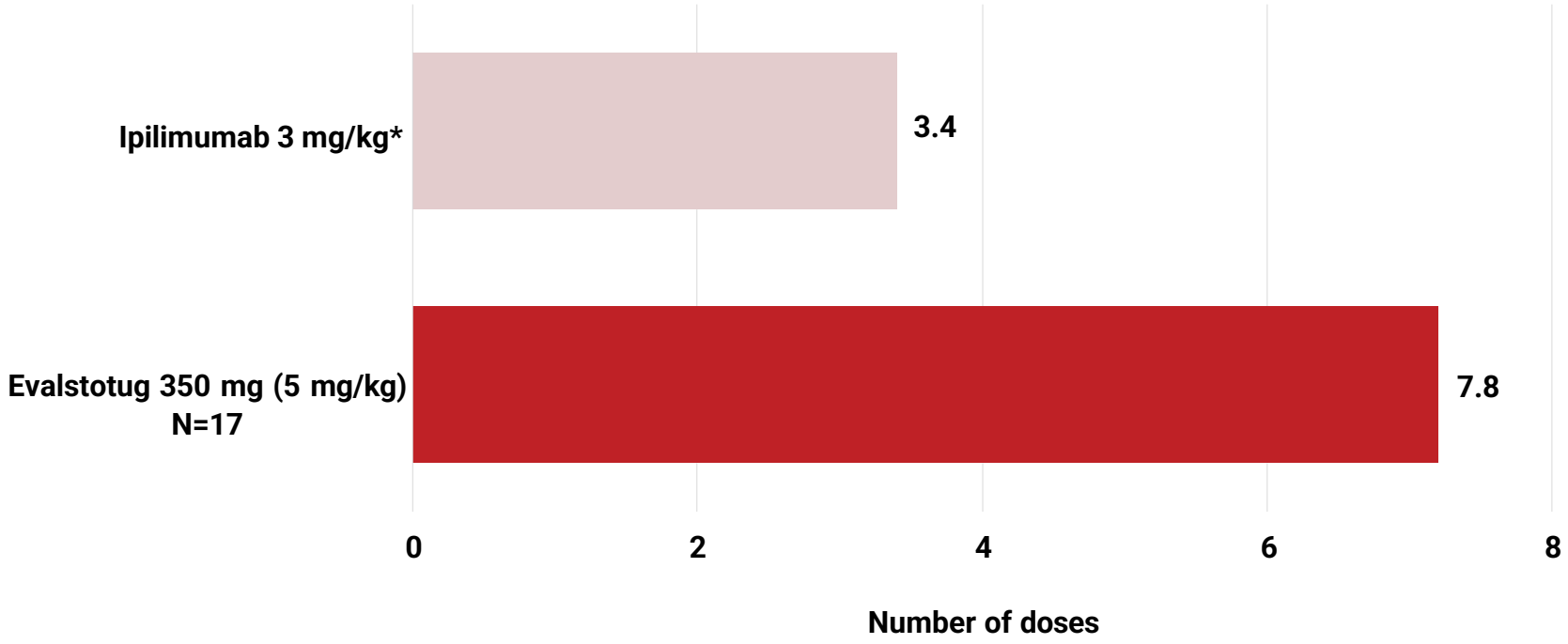
| | Total (N=17) |
|---|---|
| **Age, y, mean (SD)** | 60 (14) |
| **Sex, n (%)** | |
| Female | 8 (47) |
| Male | 9 (53) |
| **White race, n (%)** | 14 (82) |
| **ECOG, n (%)** | |
| 0 | 12 (71) |
| 1 | 5 (29) |
| **Prior Anti-PD-1 Therapy, n (%)** | 14 (82) |

| | Total (N=17) | Prior # of treatments |
|---|---|---|
| **Tumor type, n (%)** | | |
| Melanoma | 11 | 0 - 1 |
| Gastric | 2 | 3 - 5 |
| Renal cell | 1 | 3 |
| Cervical | 1 | 3 |
| NSCLC | 1 | 3 |
| aHCC | 1 | 5 |

Data Cut Date: 31Oct25

# Patients Treated with Evalstotug Received More Than Twice as Many Doses Compared with Reported Ipi Dosing

## Mean number of evalstotug 350 mg doses vs ipilimumab when in combination with PD1

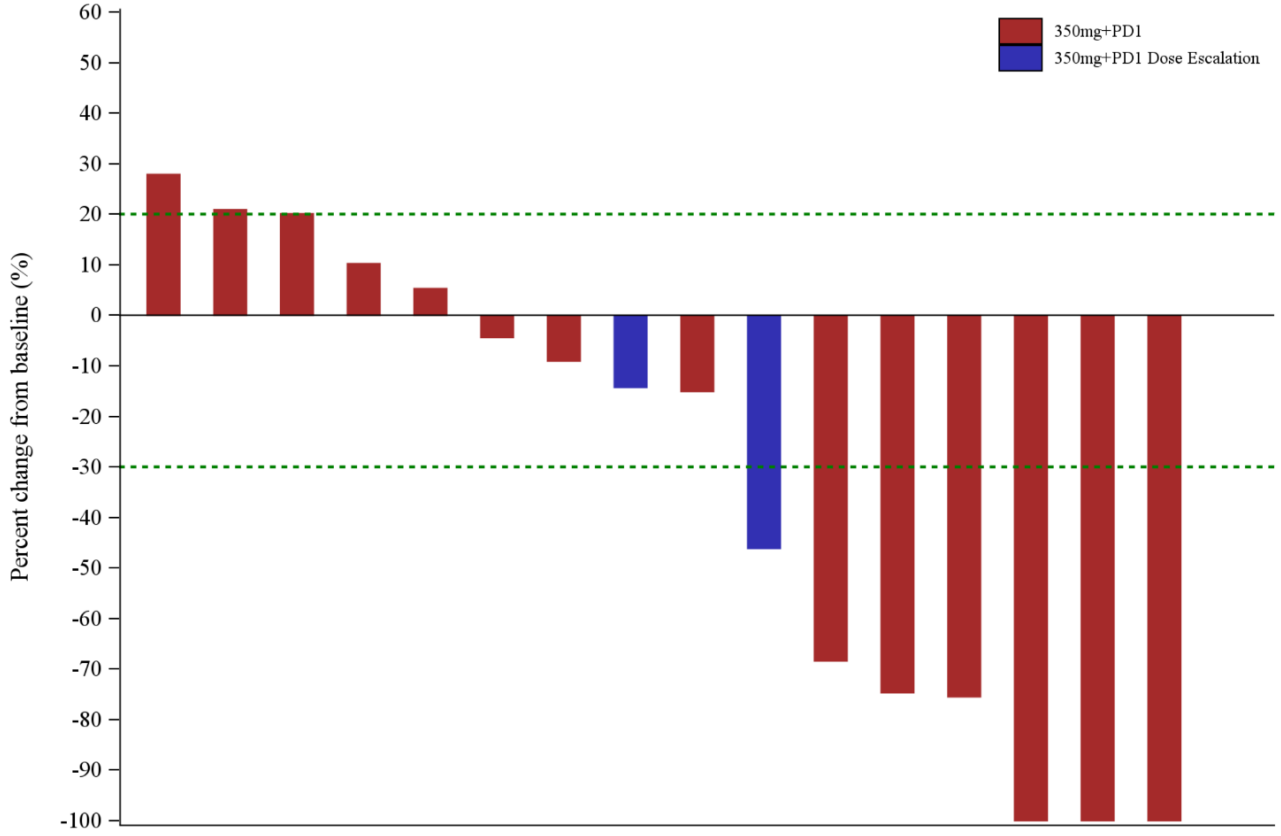



Note: Mean number of doses for ipilimumab is based on a retrospective observational study.
Evalstotug 350 mg and 700 mg are dose-equivalent to ipilimumab 5 mg/kg and 10 mg/kg, respectively.
*Mohr P, et al. J Eur Acad Dermatol Venereol. 2018;32(6):962-971.

Data Cut Date: 31Oct25

# Evalstotug at 350mg Q3W (5mg/kg based on 70 kg pt) in Combination with PD1 Across Multiple Tumor Types

## 3 Complete Responders with 2 in melanoma and 1 in cervical cancer



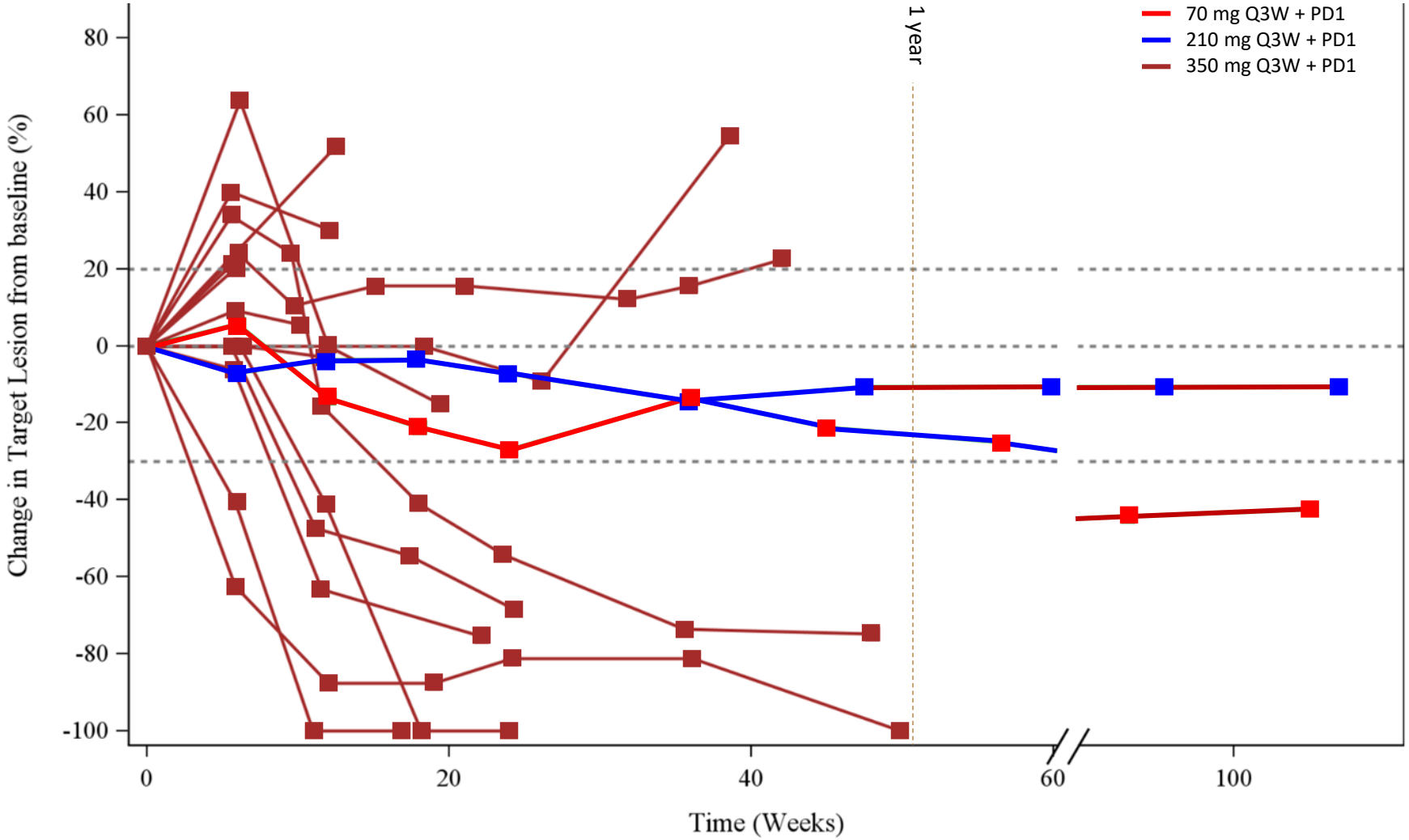

| | Total |
|---|---|
| **Responders** (confirmed & unconfirmed) | **44%** (7/17[1]) |
| **Responders** (confirmed) | **41%** (7/17[1]) |
| **DCR** | **76%** (13/17[1]) |
| **OS** | ***ongoing*** |

[1]Response evaluable patients defined as patients that had at least 1 scan after treatment with study drug

Data Cut Date: 31Oct25

# Evalstotug in Combination with PD1: 7 of 17 Achieved Response

## Durable antitumor activity across multiple solid tumor types




Data Cut Date: 31Oct25

# Evalstotug Melanoma (with or without prior ICI treatment)

# Evalstotug In Combination with PD1 in Melanoma (with or without prior ICI treatment; n=16): Demographics

## Evalstotug 350 mg or 700 mg Q3W (5 mg/kg or 10 mg/kg based on 70 kg patient)

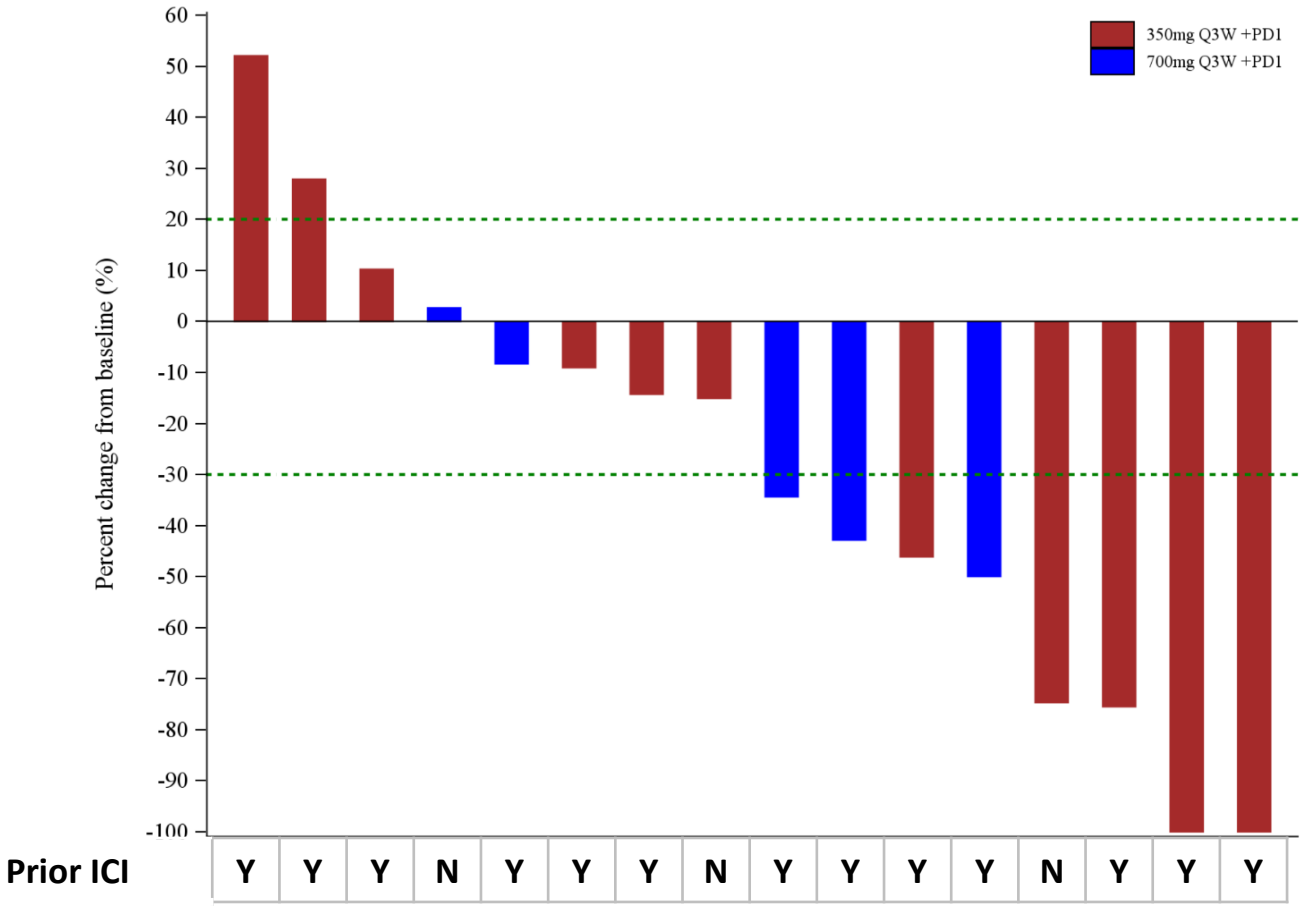
| | Total (N=16) |
|---|---|
| **Age, y, mean (SD)** | 61 (14) |
| **Sex, n (%)** | |
| Female | 10 (62) |
| Male | 6 (38) |
| **White race, n (%)** | 16 (100) |
| **ECOG, n (%)** | |
| 0 | 12 (75) |
| 1 | 4 (25) |
| **Prior ICI Therapy Status, n (%)** | |
| No prior ICI treatment | **3 (19)** |
| Adjuvant | **10 (62)** |
| Metastatic setting | **3 (19)** |

ICI = PD1 or PD1 + LAG3

Data Cut Date: 31Oct25

# Evalstotug in Combination with PD1 in Melanoma (with or without prior ICI treatment)

## 8 responders among 16 patients; 81% of patients had prior ICI



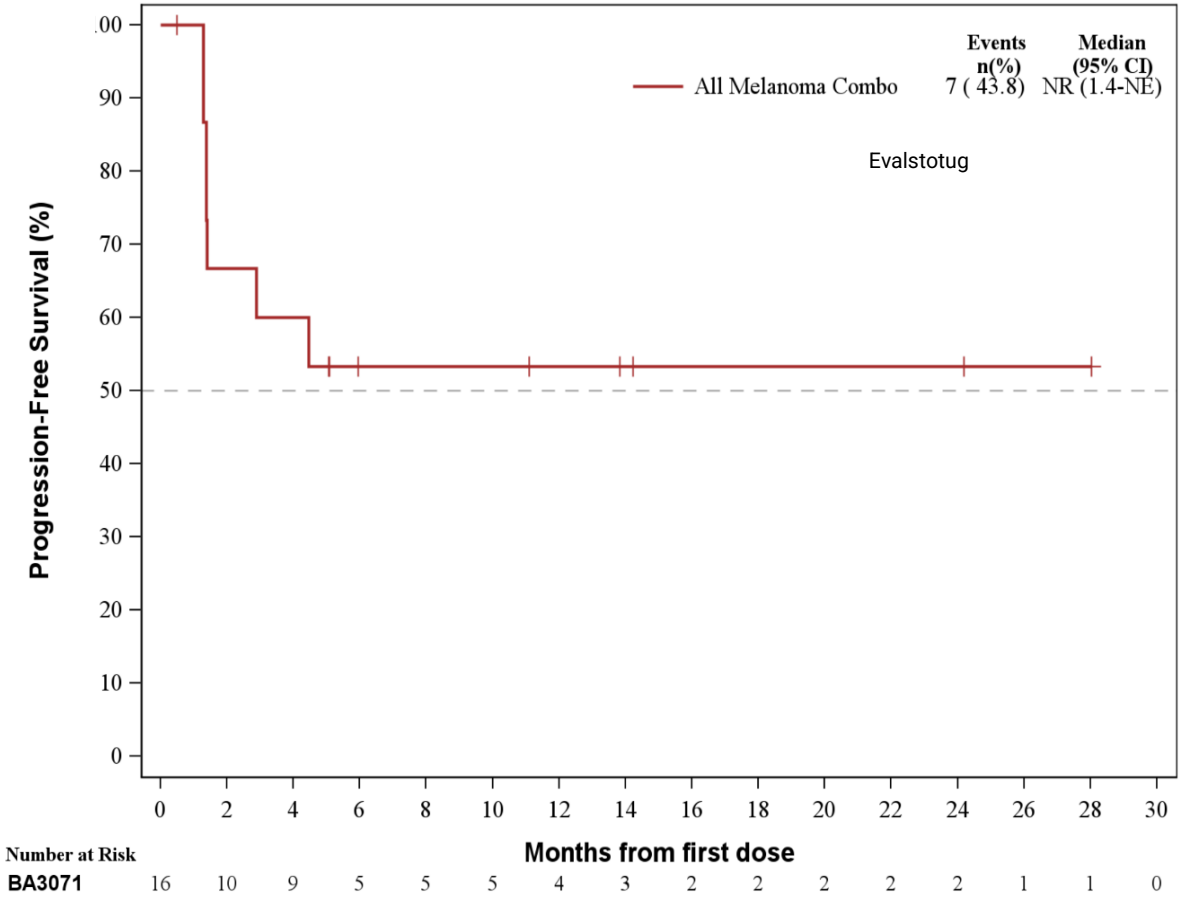

| | Total |
|---|---|
| **Responders** (confirmed & unconfirmed) | **50%** (8/16[1]) |
| **Responders** (confirmed) | **50%** (8/16[1]) |
| **DCR** | **94%** (15/16[1]) |
| **PFS** | ***Not Reached*** |
| **OS** | ***Not Reached*** |

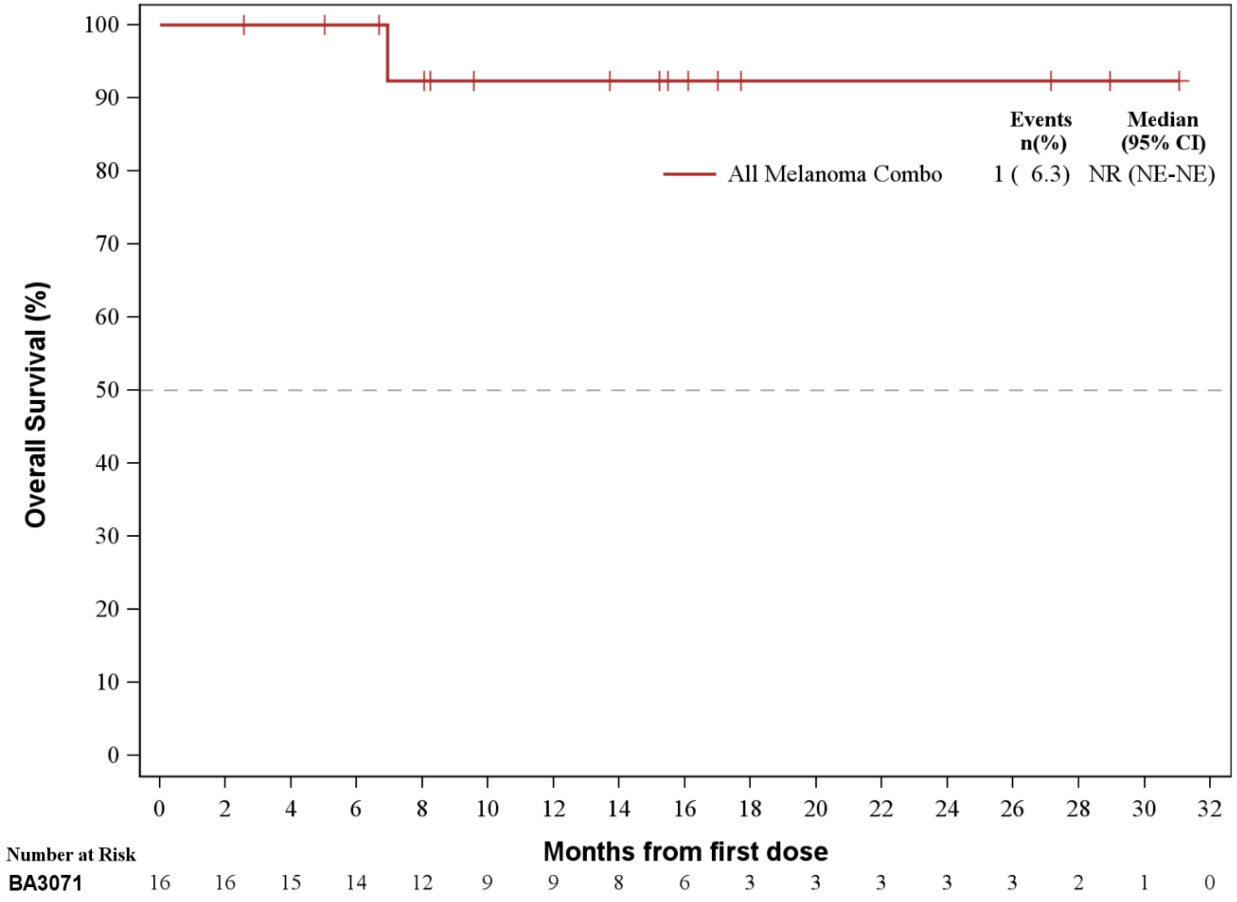
[1]Response evaluable patients defined as patients that had at least 1 scan after treatment with study drug

Data Cut Date: 31Oct25

# 50% ORR and 94% DCR in Melanoma (with or without prior ICI treatment)

## 81% patients had received prior PD1



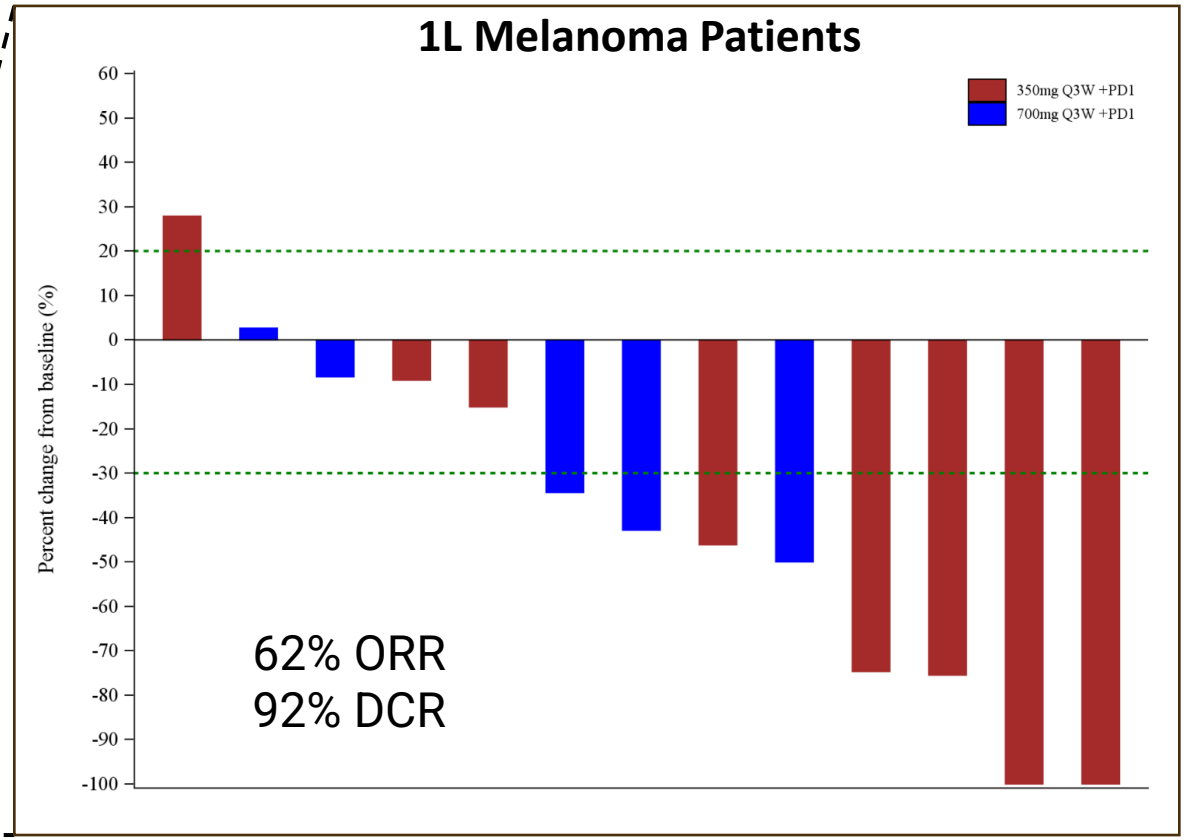

Data Cut Date: 31Oct25

# Evalstotug 1L Melanoma – prior adjuvant therapy or no prior ICI treatment

# Evalstotug In Combination with PD1 in Melanoma (no prior treatment or prior adjuvant therapy; n=13): Demographics and Waterfall

## Evalstotug 350 mg or 700 mg Q3W (5 mg/kg or 10 mg/kg based on 70 kg patient)

| | Total (N=16) |
|---|---|
| **Age, y, mean (SD)** | 61 (14) |
| **Sex, n (%)** | |
| Female | 10 (62) |
| Male | 6 (38) |
| **White race, n (%)** | 16 (100) |
| **ECOG, n (%)** | |
| 0 | 12 (75) |
| 1 | 4 (25) |
| **Prior ICI Therapy Status, n (%)** | |
| No prior ICI treatment | **3 (19)** |
| Adjuvant | **10 (62)** |
| Metastatic setting | **3 (19)** |

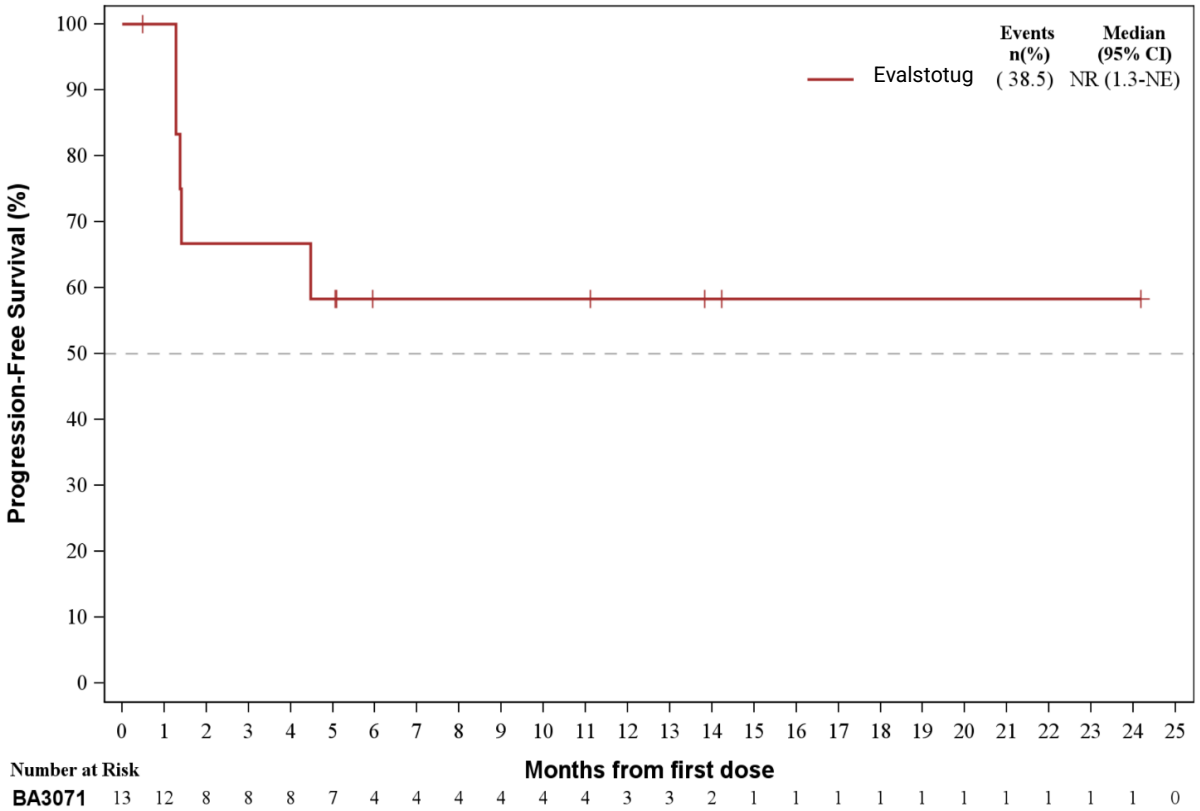


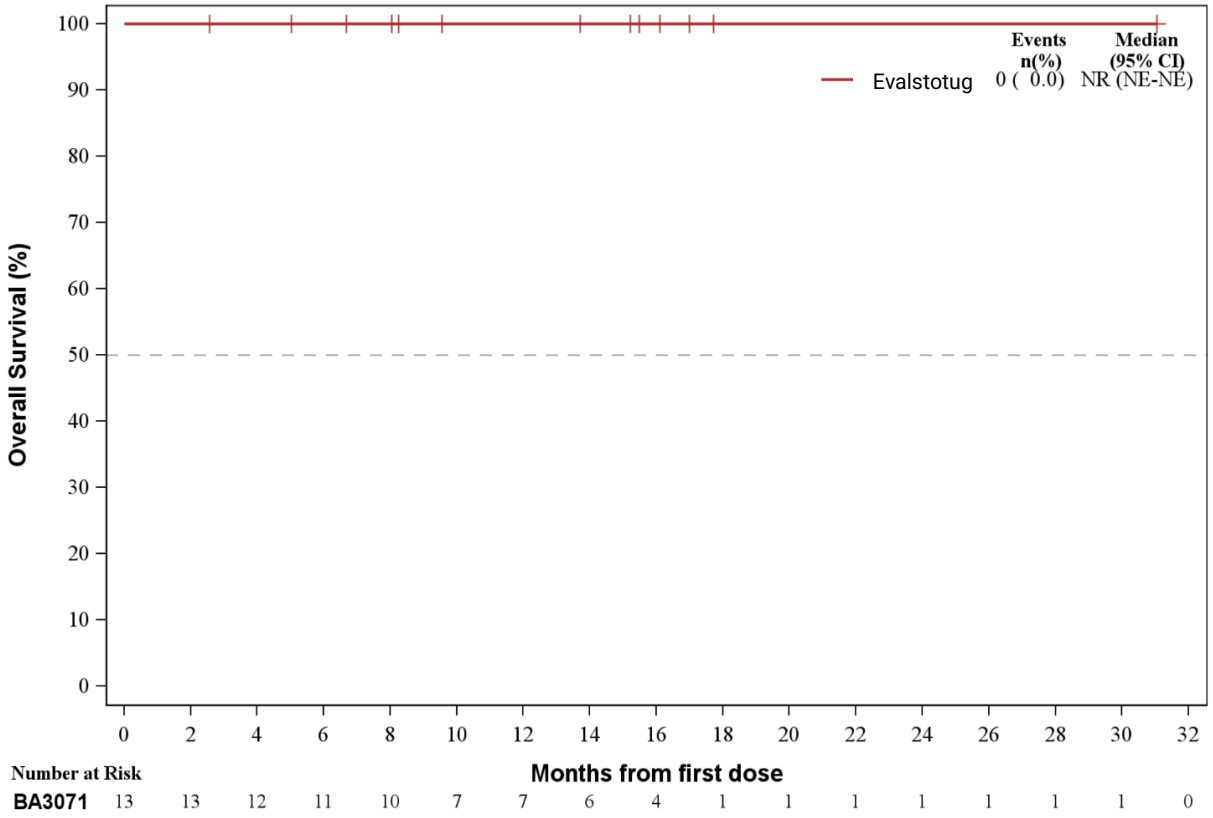

ICI = PD1 or PD1 + LAG3

Data Cut Date: 31Oct25

# 62% ORR and 92% DCR in Melanoma (with or without prior ICI treatment; n=13) Evalstotug in Combination with PD1

## 81% patients had received prior PD1 adjuvant treatment



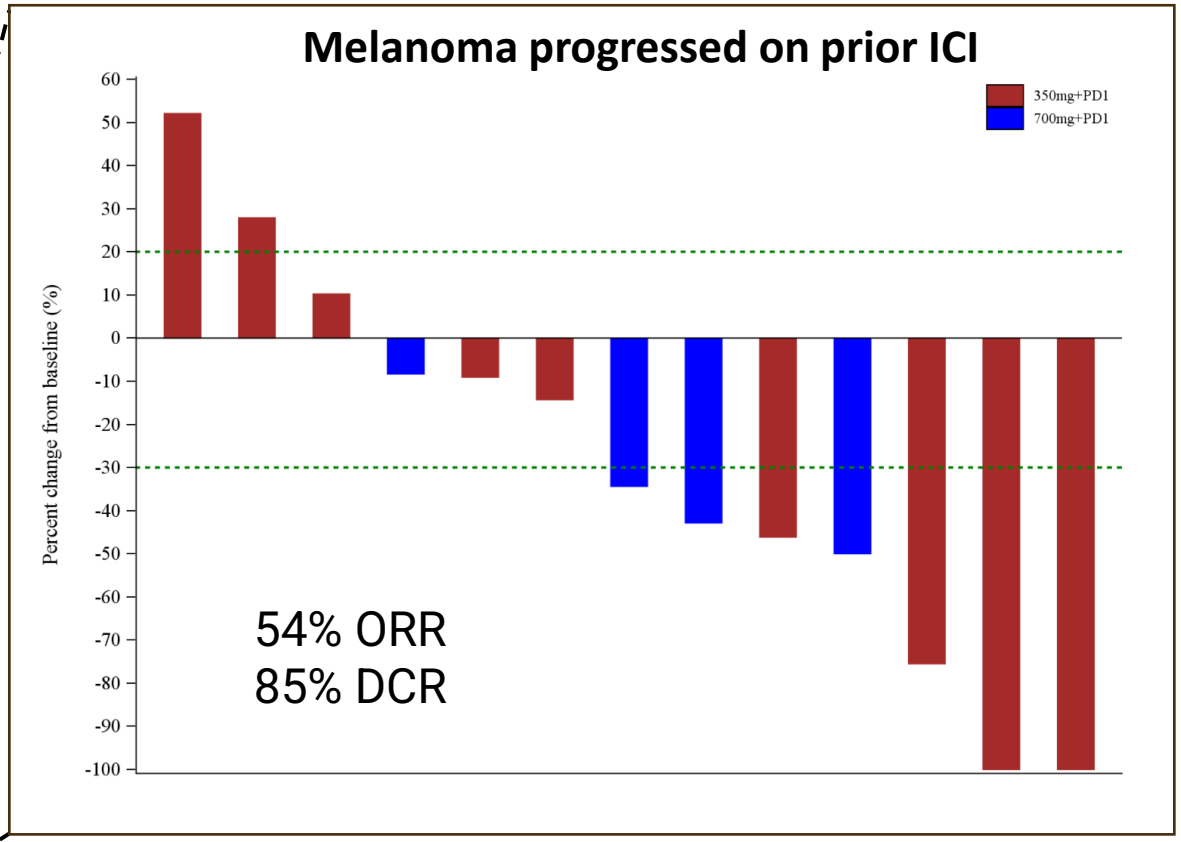

Data Cut Date: 31Oct25

# Evalstotug 1L and 2L+ Melanoma who received prior ICI treatment

# Evalstotug In Combination with PD1 in Melanoma (received prior ICI treatment; n=13): Demographics and Waterfall

## Evalstotug 350 mg or 700 mg Q3W (5 mg/kg to 10 mg/kg based on 70 kg patient)

| | Total (N=16) |
|---|---|
| **Age, y, mean (SD)** | 61 (14) |
| **Sex, n (%)** | |
| Female | 10 (62) |
| Male | 6 (38) |
| **White race, n (%)** | 16 (100) |
| **ECOG, n (%)** | |
| 0 | 12 (75) |
| 1 | 4 (25) |
| **Prior ICI Therapy Status, n (%)** | |
| No prior ICI treatment | **3 (19)** |
| Adjuvant | **10 (62)** |
| Metastatic setting | **3 (19)** |

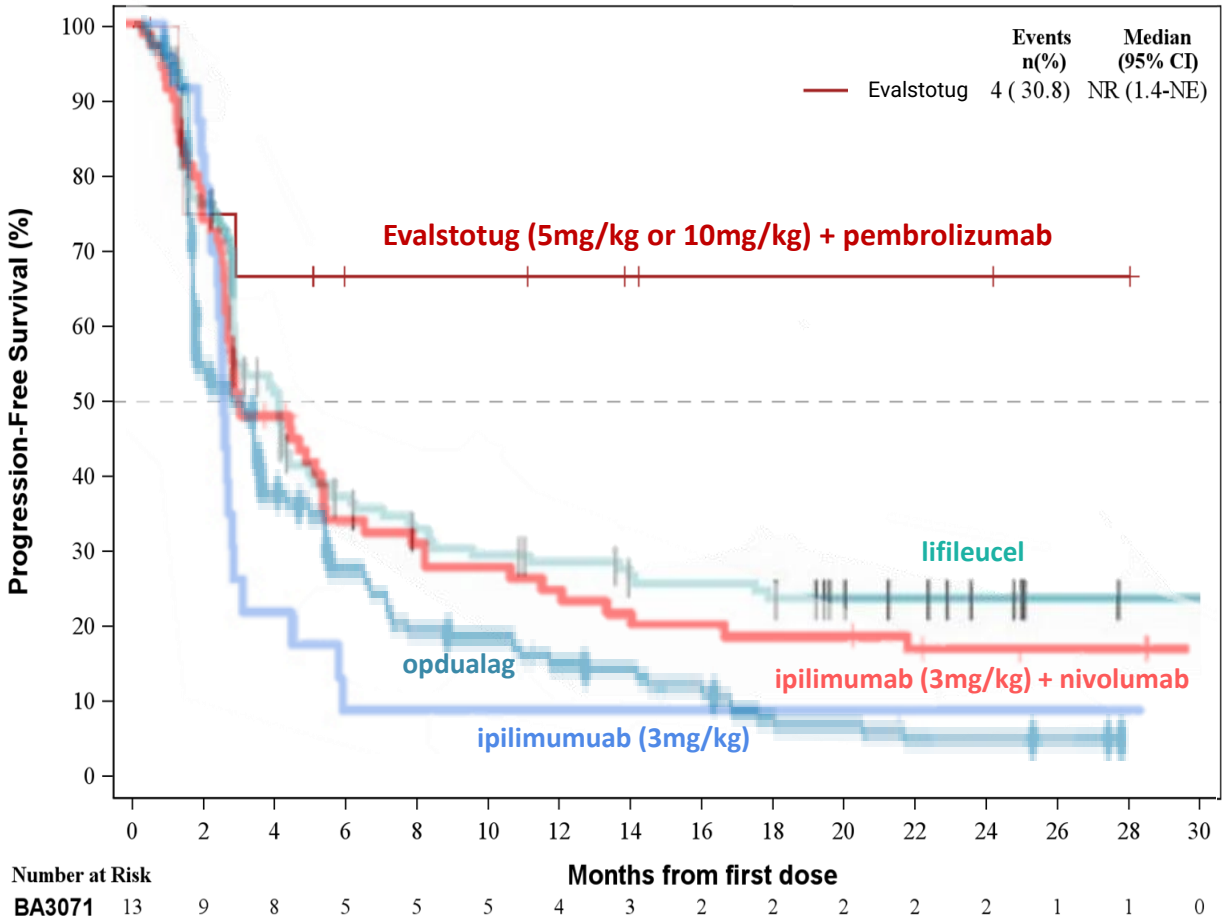


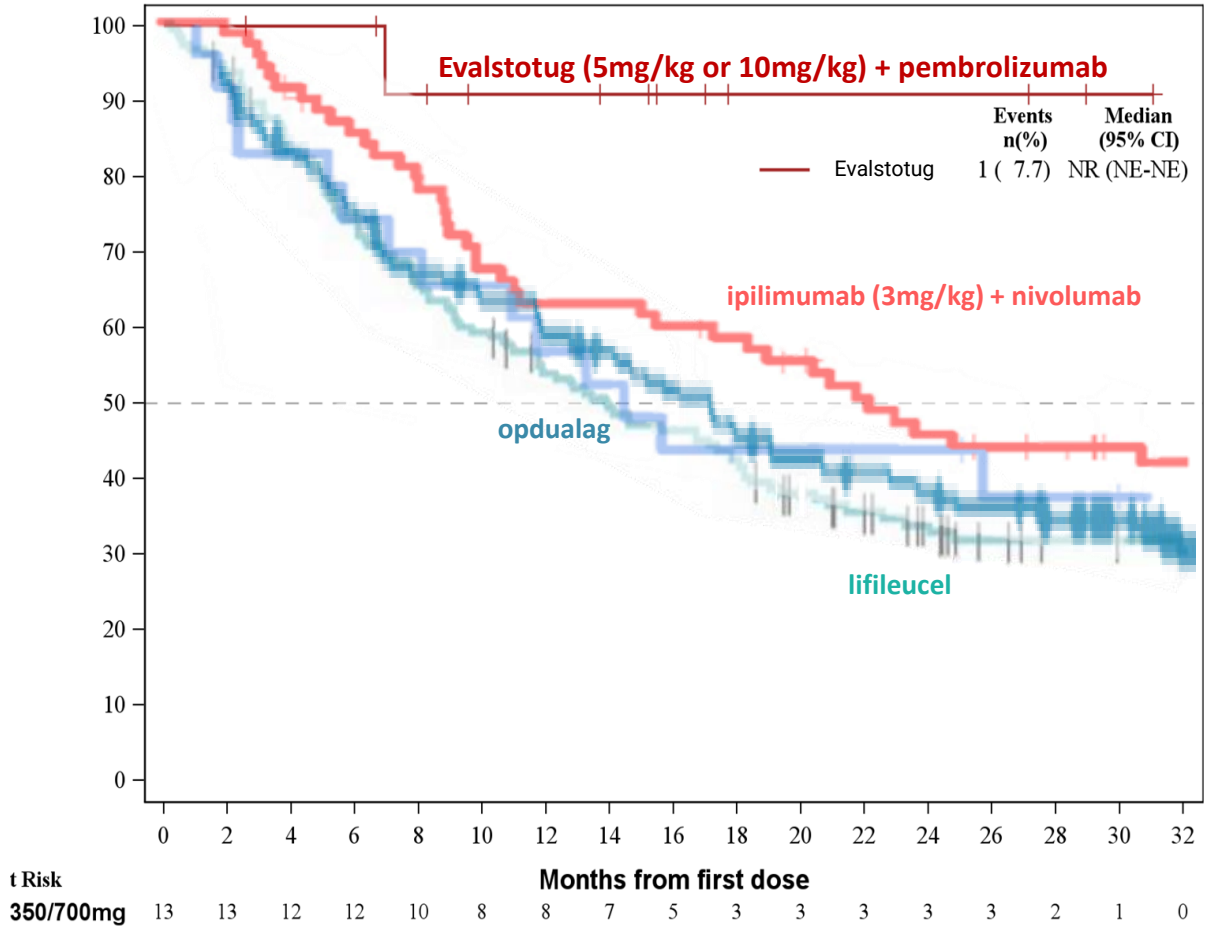

ICI = PD1 or PD1 + LAG3

Data Cut Date: 31Oct25

# 54% ORR and 85% DCR in 1L and 2L+ Melanoma who received Prior ICI

## All 13 patients received prior ICI; evalstotug at higher doses achieves greater efficacy




Data Cut Date: 31Oct25

# Safety

# Evalstotug vs Ipilimumab: Cross Trial Comparison

## Lower imAE rate vs Ipilimumab despite more patients previously treated with ICI

| Treatment | Evalstotug (5 mg/kg) + PD1 Q3W N=17 | Evalstotug (5 mg/kg or 10 mg/kg) + PD1 Q3W N=25 | Ipilimumab (3 mg/kg) + nivolumab Q3W N=178-314[1,2,3] |
|---|---|---|---|
| Doses | 1 – ≤18 weeks exposure (≤6 doses) | | 1 – ≤12 weeks exposure (≤4 doses) |
| Tumor Types | Multiple tumor types | | Melanoma |
| % Patients w/ Prior Tx | 90% | | 15%[4] |
| imAE (G3-4) | 12% (no G4) | 16% (no G4) | 40% |

*[1]Wolchok, J; Five-Year Survival with Cmbined Nivolumab and Ipilimumab in Advanced Melanoma; N Engl J Med 2019;381:1535-1546; [2]Lebbe, C; Evaluation of Two Dosing Regimens for Nivolumab in Combination With Ipilimumab in Patients With Advanced Melanoma: Results From the Phase IIIb/IV CheckMate 511 Trial; J Clin Oncol. 2019 Feb 27;37(11):867–875; [3]Larkin, J. Combined Nivolumab and Ipilimumab or Monotherapy in Untreated Melanoma; N Engl J Med 2015;373:23-34; [4]Allouchery, M; Safety of immune checkpoint inhibitor rechallenge after discontinuation for grade ≥2 immune-related adverse events in patients with cancer; J Immunother Cancer. 2020 Dec;8(2):e001622.*

Data Cut Date: 31Oct25

# Evalstotug in Combination with PD1: Overall Safety Summary

## Generally well-tolerated; All G3 and G4 resolved

| Related AE Summary | Total (N=17) | Total (N=8) | Total (N=25) |
|---|---|---|---|
| Any Related Adverse Events (AEs) | 16 (94%) | 8 (100%) | 24 (96%) |
| Related AEs with CTCAE[1] | | | |
| Grade 3[2] | 6 (36%) | 4 (50%) | 12 (48%) |
| Grade 4[2] transient hypercalcemia | 1 (6%) | 1 (13%) | 8 (32%) |
| imAE Grade 3 or 4 | 5 (29%) | 2 (25%) | 7 (28%) |
| Any related serious AEs[2] | 5 (29%) | 5 (63%) | 10 (40%) |
| Possibly Related AEs leading to death[2] | 0 | 0 | 0 |
| Related AEs leading to treatment discontinuation[2] | 5 (29%) | 3 (38%) | 7 (28%) |

[1]CTCAE: Common Terminology Criteria for Adverse Events. The NCI Common Terminology Criteria for Adverse Events is a descriptive terminology which is utilized for Adverse Event (AE) reporting. A grading (severity) scale is provided for each AE term.
[2]As assessed by the investigator. Missing responses are counted as related.

Data Cut Date: 31Oct25

# Evalstotug in Combination with PD1 Safety Data

## Treatment-emergent related adverse events >15%

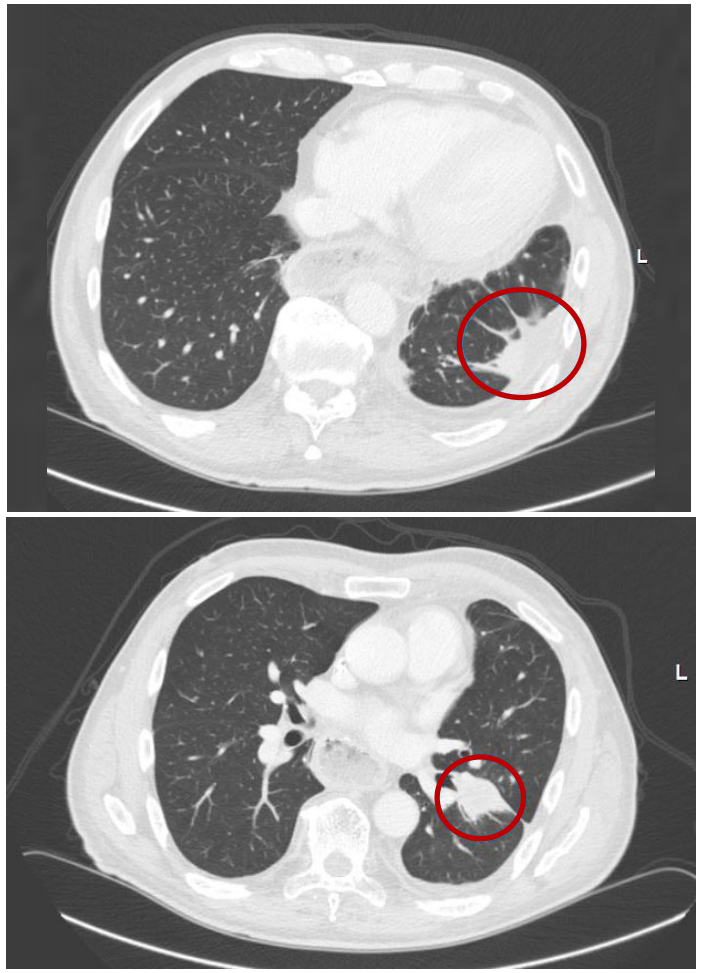
| | Evalstotug 350 mg + PD1 (N=17) | | Evalstotug 700 mg + PD1 (N=8) | | Total (N=25) | |
|---|---|---|---|---|---|---|
| AE | All | Gr3+ | All | Gr3+ | All | Gr3+ |
| Chills | 11 (64.7) | 0 | 4 (50.0) | 0 | 15 (60.0) | 0 |
| Fatigue | 11 (64.7) | 0 | 3 (37.5) | 1 (12.5) | 14 (56.0) | 1 (4.0) |
| Nausea | 8 (47.1) | 0 | 4 (50.0) | 0 | 12 (48.0) | 0 |
| Cytokine release syndrome | 6 (35.3) | 0 | 5 (62.5) | 0 | 11 (44.0) | 0 |
| Vomiting | 9 (52.9) | 0 | 2 (25.0) | 0 | 11 (44.0) | 0 |
| Arthralgia | 9 (52.9) | 0 | 1 (12.5) | 1 (12.5) | 10 (40.0) | 1 (4.0) |
| Diarrhoea | 6 (35.3) | 1 (5.9) | 3 (37.5) | 0 | 9 (36.0) | 1 (4.0) |
| Abdominal pain | 6 (35.3) | 1 (5.9) | 2 (25.0) | 0 | 8 (32.0) | 1 (4.0) |
| Pruritus | 4 (23.5) | 0 | 4 (50.0) | 0 | 8 (32.0) | 0 |
| Rash | 6 (35.3) | 0 | 2 (25.0) | 0 | 8 (32.0) | 0 |
| Anaemia | 5 (29.4) | 3 (17.6) | 2 (25.0) | 0 | 7 (28.0) | 3 (12.0) |
| Headache | 6 (35.3) | 0 | 1 (12.5) | 0 | 7 (28.0) | 0 |
| Oedema peripheral | 3 (17.6) | 0 | 3 (37.5) | 0 | 6 (24.0) | 0 |
| Hypokalaemia | 4 (23.5) | 1 (5.9) | 1 (12.5) | 0 | 5 (20.0) | 1 (4.0) |
| Decreased appetite | 4 (23.5) | 0 | 1 (12.5) | 0 | 5 (20.0) | 0 |
| Infusion related reaction | 2 (11.8) | 0 | 3 (37.5) | 0 | 5 (20.0) | 0 |
| Lipase increased | 3 (17.6) | 2 (11.8) | 1 (12.5) | 0 | 4 (16.0) | 2 (8.0) |
| Pneumonia | 1 (5.9) | 0 | 3 (37.5) | 2 (25.0) | 4 (16.0) | 2 (8.0) |
| Dehydration | 3 (17.6) | 1 (5.9) | 1 (12.5) | 0 | 4 (16.0) | 1 (4.0) |
| Back pain | 3 (17.6) | 0 | 1 (12.5) | 0 | 4 (16.0) | 0 |
| Cough | 4 (23.5) | 0 | 0 | 0 | 4 (16.0) | 0 |
| Dyspnoea | 3 (17.6) | 0 | 1 (12.5) | 0 | 4 (16.0) | 0 |
| Hypomagnesaemia | 3 (17.6) | 0 | 1 (12.5) | 0 | 4 (16.0) | 0 |
| Influenza like illness | 4 (23.5) | 0 | 0 | 0 | 4 (16.0) | 0 |
| Pyrexia | 3 (17.6) | 0 | 1 (12.5) | 0 | 4 (16.0) | 0 |

Data Cut Date: 31Oct25

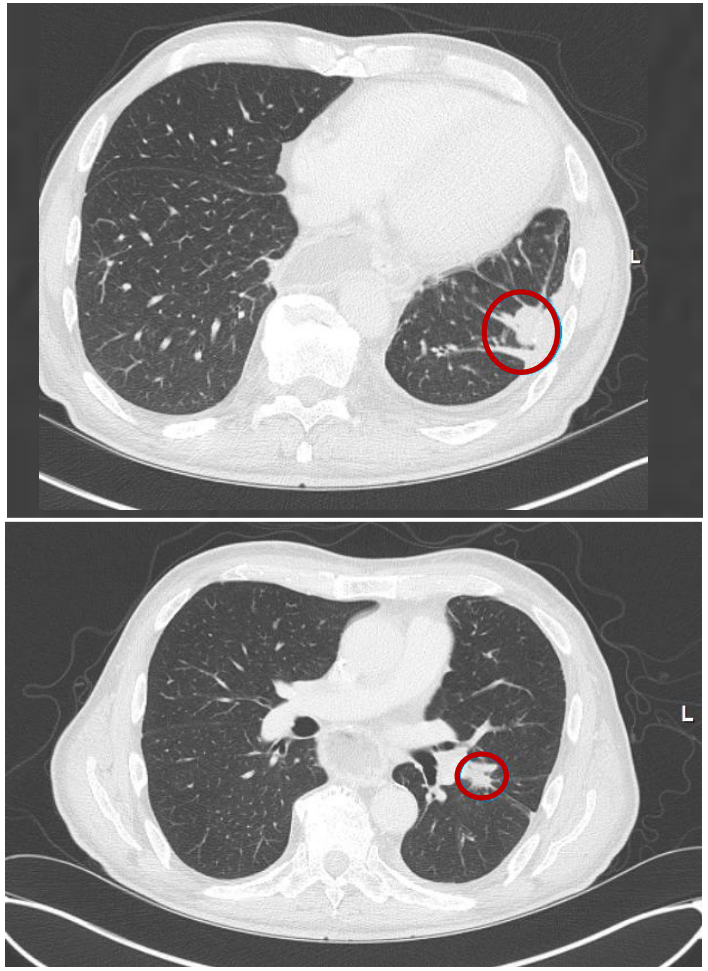
# Confirmed PR - Gastro-esophageal Cancer

## 63-year-old male, stage IV gastro-esophageal cancer HER2 negative, post-FOLFOX, taxane, TKI, anti-PD1 and anti-VEGFI

Baseline - July 31,2023



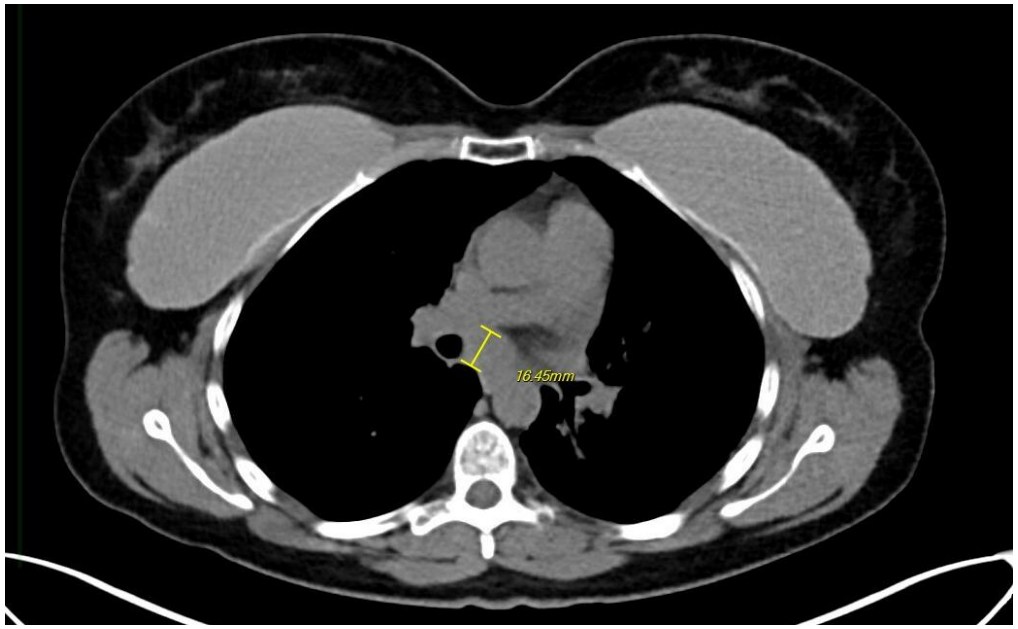
On Treatment - October 23, 2023



# Confirmed CR - Cervical Cancer

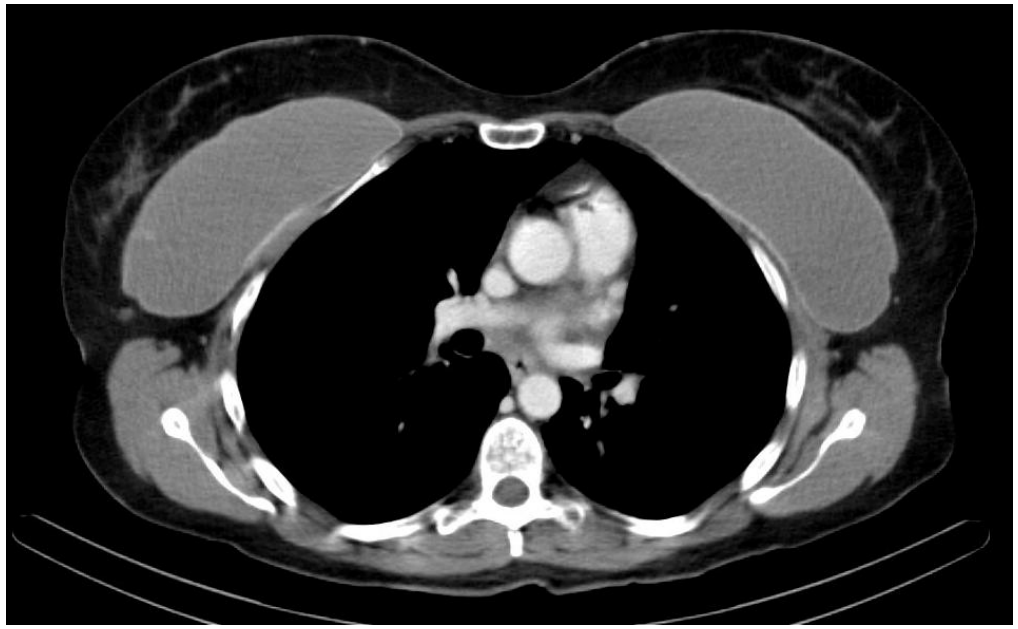
## 43-year-old female, stage IV cervical cancer HPV+16 positive, post-platinum, taxane, anti-PD1 and anti-VEGF

**Baseline – March 23, 2023**




"Multiple enlarged mediastinal, paraesophageal, and right hilar lymph nodes…"

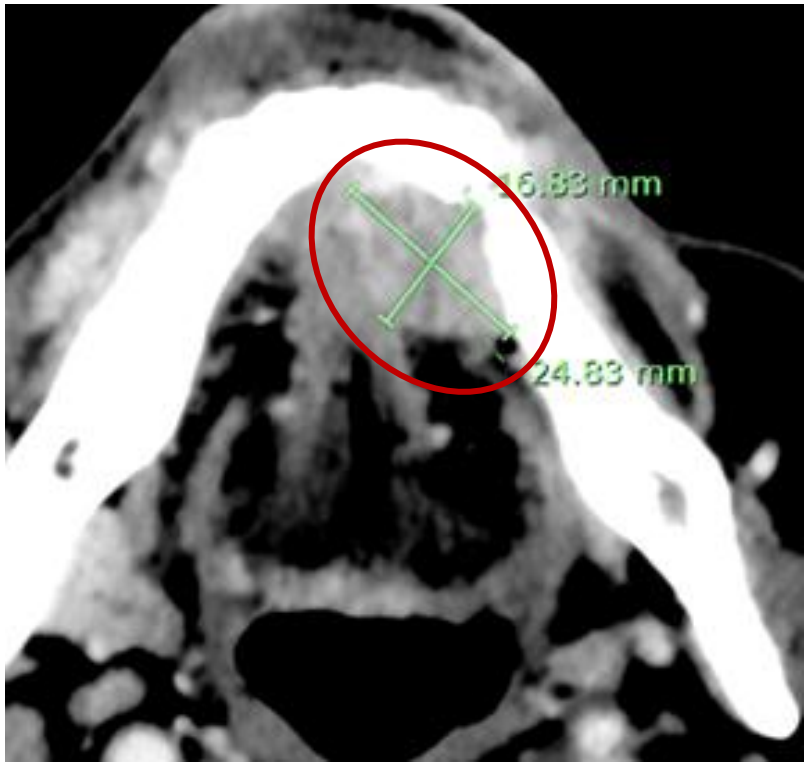
**On Treatment – August 9, 2023**

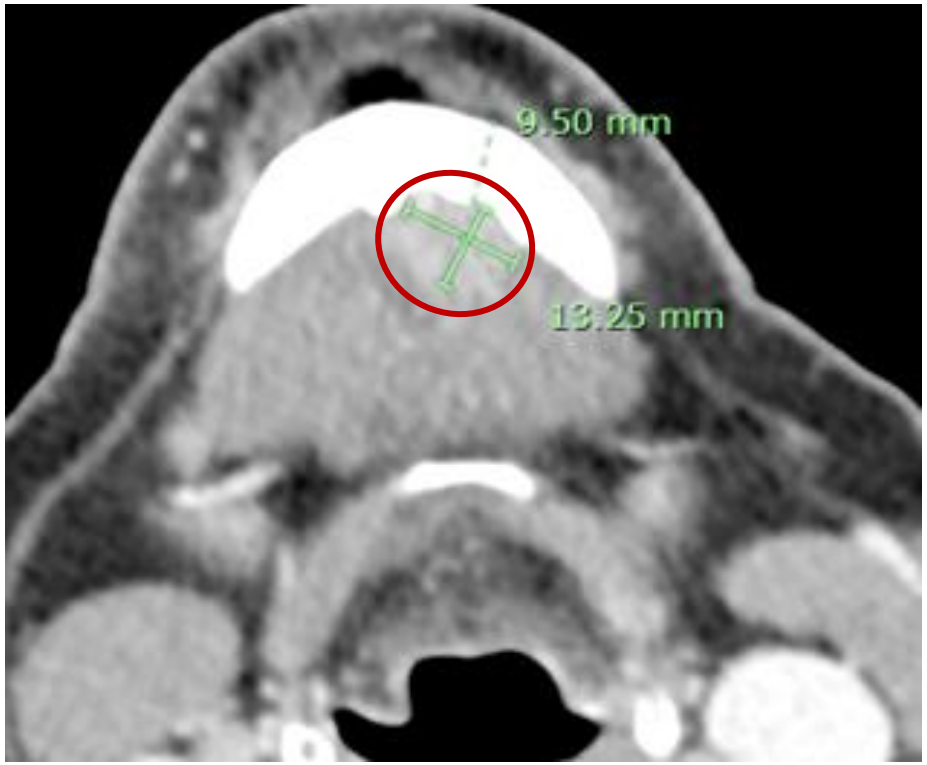


"No enlarged mediastinal, hilar or axillary lymph nodes are present. There is persistent resolution of previously noted enlarged mediastinal and paraesophageal lymph nodes."

# Evalstotug Enables the Combination with ADC and PD1 Antibodies
# PR (-50%) – Well Tolerated Triplet; Oz-V + Evalstotug + PD1

## 69 yo M with oral cavity, floor of mouth/ mandibular, mucoepidermoid carcinoma; neo-adjuvant patient




Disease had recurred after prior surgery and chemoradiation (platinum / taxane / pembrolizumab as well as cetuximab). Prior to Oz-V triplet patient had tumor filling the maxillary sinus measuring 5.3cm in longest diameter. After triplet, the tumor nearly resolved and is difficult to measure. A transient, clinically asymptomatic elevation of hepatic transaminases was documented that didn't recur with subsequent dosing.

# FDA Guidance Regarding Evalstotug Pivotal Trial in 1L Unresectable and / or Metastatic Melanoma

- Centrally reviewed PFS acceptable as primary endpoint
- General agreement with proposed study population and sample size
- Additional guidance received on ongoing dose optimization and control arm:
  - IO-based combination regimen should be included in the control arm
  - Project Optimus should guide determination of Phase 3 evalstotug dose

# Overall Summary: Evalstotug + PD1

## Greater efficacy and lower imAE rate vs ipilimumab + PD1

- Preclinical & clinical data demonstrate that
  - ipi & evalstotug are similar, *i.e.* epitope, affinity, $T_{1/2}$ and tumor exposure, and efficacy
  - ipi & evalstotug are NOT similar with respect to normal tissue environment and safety; *e.g.*, reduced imAEs and extended treatment
- 54% ORR and 85% DCR in 1L and 2L+ melanoma patients who received prior ICI treatment
- Higher dosing yields encouraging efficacy with low incidence and severity of imAEs, consistent with CAB-driven tumor selectivity for potential best-in-class CTLA4
- Significant opportunity for an effective and better tolerated CTLA-4 in combination regimens

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